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A Comparative Assessment of Environmental Problems. Public Health Risk Work Group Report

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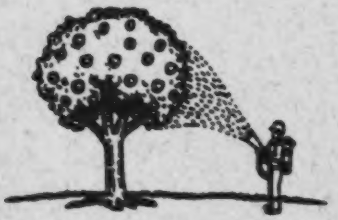
Unfinished Business in New England: A Comparative Assessment of Environmental Problems

Public Health Risk Work Group Report



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This report presents the results of a study undertaken by EPA in New England that examined 24 environmental problems and assessed the level of residual risk posed by each problem to human health. Using the methodological framework of risk assessment the public health work group developed estimates of residual risk for individuals and populations for each of the problem areas. Residual risk is defined as the risk posed by a problem, given current levels of control. Analyses for cancer risk and non-cancer risk were developed independently and then combined at the end of the study to produce a single ranking. The Public Health Risk Work Group Report of Unfinished Business in New England: A Comparative Assessment of Environmental Problems found that ozone, radon and lead pose the highest level of residual risk to human health in New England.

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Public Health Risk Work Group Report

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I. Introduction

The Public Health Risk Work Group was charged with evaluating the residual cancer and non-cancer risks posed by the 24 environmental problem areas being addressed in the Risk Reduction Project (RRP) and then providing a relative ranking of residual public health risk. The work group's initial goal was to perform a quantitative analysis for each problem area. However, since available data were often limited and not easy to compare directly, we relied on a significant amount of professional judgment during the ranking process. The conclusions reached by the work group therefore represent judgments and not verifiable fact. Nevertheless, the work group feels confident that the rankings we produced represent relative differences in residual public health risk across environmental problem areas.

The work group was composed of technical staff from each of the Region I program offices and was supported by a representative from New England interstate environmental agencies, a representative from the EPA Headquarter's Office of Policy, Planning and Evaluation, and a contractor. Representatives were selected based on public health expertise and specific program knowledge. The work group first discussed the methodology to be used in assessing residual public health risk. Group members were then designated as problem area leads according to their individual areas of expertise. Work group members, or contractors under their direction, then prepared "plan of analysis" papers outlining how each problem area would be analyzed to determine the residual risk it posed. The plans were then discussed by the work group. With contractor assistance, the work group members then collected and analyzed relevant data and prepared their analyses for consideration by the rest of the work group. The work group met twice each month over a period of four months to develop its methods and outline the problem areas, and then weekly for one month to discuss the evaluation of each problem area and perform the ranking.

This report first discusses the methodology developed by the Public Health Risk Work Group to evaluate the problem areas. We present our general approach and then discuss the specifics of the cancer and non-cancer approaches. The next section presents the ranking process for cancer and non-cancer effects, followed by the process used to generate the overall public health risk ranking. A final section provides our observations on the difficulties we encountered and other key lessons we learned in the process. The appendix to this report presents in greater detail the methods, data, and conclusions for each of the problem areas.

II. Analytical Methodology

Since there is no single, EPA-endorsed, off-the-shelf method for conducting a comparative public health risk evaluation, the work group set out to define a process to govern our analysis and deliberations. This section describes the work group's ground rules, the general method we developed, and more specific details on the cancer and non-cancer methodologies.

A. General Approach

Ground Rules

At the outset of the project certain ground rules were established to provide consistency across the three work groups participating in the RRP. Two of these ground rules were very important to the way the Public Health Risk Work Group approached the analysis. First, we considered each problem area assuming current levels of control and compliance. This means we considered the residual risk posed by the problem area and not the risk that might be posed if controls were changed or if compliance rates dropped.

This led to a discussion of what constitutes controls. The work group expanded the obvious list of pollution control equipment and pre-treatment to include fencing of Superfund sites or other waste facilities. We recognize that trespass can and will occur and have evaluated that risk, but we have assumed that one cannot currently drill a drinking water well on-site at such a facility. We did not include announced "advisories" restricting fishing or swimming as controls since they depend on the awareness and cooperation of the public for effectiveness.

The second important ground rule was that we consider current emissions and their effects now and in the future. We sought the most recent data available to define those current emissions with the understanding that data as much as three to five years old might be the best available. In cases where effects happen over many years, we did not discount the future effects.

Health Risk Criteria

With the ground rules set, we next considered the types of public health risk to analyze. We decided to first look at cancer and non-cancer risks separately, postponing until later a discussion on how we would combine the two. Within these two categories we agreed to examine both individual risk and population risk. Individual risk was defined as the risk posed to an individual potentially exposed to the chemicals under consideration at what was determined to be an average concentration. Population risk considered the risk posed to all people determined to be exposed to this concentration in Region I. In both the individual and population risk analyses, we agreed to consider realistic, average exposures, not worst-case exposures. Thus we have not explicitly considered the maximum exposed individual. Finally, we agreed to consider both chronic and acute effects, focusing on the more typical exposure for each problem area.

Overall Methodology

We then developed an overall methodology for assessing the types of public health risks described above. In developing the methodology, we used the approach taken in the National Comparative Risk Project (NCRP) as the point of departure and the considered alternatives at each step. While our method ended up looking quite similar to the NCRP method, we did not take it as a given at the outset. The broad outline of our methodology is described below, with further details on the cancer and non-cancer approaches presented in succeeding sections.

The first step was to identify a short list of chemicals and the associated possible exposure pathways to represent each problem area. We relied on individual expertise to identify the chemicals, sources, and pathways. The chemicals were selected on the basis of how well they reasonably characterized the problem and the extent to which data were available. The number of chemicals studied for any problem area ranged from 1 (e.g., lead, asbestos, radon) to 10.

The second step was to characterize the potency or dose-response relationship for each chemical. While this characterization was straightforward in the cancer analysis, developing an analogous approach for non-carcinogens was complicated, and the reader is referred to the non-cancer methods section for further details.

To characterize the severity of the effects, we added an additional step to the non-cancer methodology. This was necessary because, unlike cancer, there are many different types of non-cancer effects, and they vary widely in severity from one chemical to the next. These systemic toxic effects include cardiovascular, developmental, immunological, kidney, liver, gastrointestinal, neurotoxic/behavioral, reproductive, respiratory, and other effects. The work group embraced the NCRP approach of using a severity index to sort out the type of impairment and the part of the body affected. Further details are provided in the non-cancer methods section below.

The third step was to assess exposures. This involved determining the likely concentration levels at which exposure occurs, the resulting doses, and the likely population exposed. We focused on average rather than worst-case exposures. The exposure assessment was based on available ambient monitoring data, modeled estimates, and available data on actual incidence. The method for any problem area depended on the availability of actual data or an appropriate modeling approach. Rather than selecting one method, the work group sought the best available exposure estimate within each problem area.

The next step was to combine these factors to characterize the individual and population risk for each chemical within each problem area. In the cancer analysis we quantified individual risk and a simplified estimate of annual cancer incidence. In the non-cancer analysis we developed a combined score based on the exposure, potency, and severity scores. In problem areas with more than one chemical, the risks were combined to arrive at an overall cancer risk, while for non-cancer risk the highest score among all chemicals was used.

The final step in the process was to develop estimates of the percentage of the problem area covered and the uncertainty of our risk estimate. These were described qualitatively as high, medium, or low. The estimate on percentage of problem covered was intended to help us account for the portion of the issue not analyzed when it came time to rank the problem areas. We decided not to use the estimate of the problem area covered to mathematically scale the risk estimate. Rather, we used the estimate to provide a qualitative basis for adjusting the risk estimate relative to other problem areas. The uncertainty estimate was used to characterize our confidence in the data. We used best-guess estimates of uncertainty to help provide some bounding of our risk estimate.

Other Considerations

The work group decided to refine the list of environmental problem areas. First, we eliminated two of the problem areas from further analysis. We eliminated Wetlands/Habitat Loss (#11), since there clearly were no direct public health effects associated with this problem area. We also eliminated Lakes, Ponds, and Impoundments (#24), since we could not readily distinguish any public health risks not already covered by Discharges to Surface Waters or Estuaries (problem areas #7 through #10).

Discharges to Surface Waters from Industrial Point Sources (problem area #7), from Publicly Owned Treatment Works (POTWs) (problem area #8), and from Nonpoint Sources (problem area #9) were analyzed as a group due to our inability at the beginning of the analysis to separate out the contributions of each problem area to the water quality data collected. Once the risks were characterized and the chemicals posing the greatest risks were identified, an attempt was made to distribute the risks across the three problem areas.

We also refined some of the problem area definitions to make more explicit what was or was not included and to reduce double-counting as much as possible. For instance, we decided that radon and lead exposures through drinking water would be covered in the Radon (#4) and Lead (#22) problem areas, respectively, and not under Drinking Water (#12). We included effects from the incineration of most hazardous waste under RCRA Waste Sites (#13), while we considered combustion of waste oil under Hazardous/Toxic Air Pollutants (#3). We included asbestos exposures under the Asbestos (#23) problem area rather than under Indoor Air Pollutants Other Than Radon (#5). Other, less substantial, refinements to the definitions are described in the papers for each individual problem area found in the appendix.

B. Cancer Methodology

This section describes the cancer methodology in a step-by-step fashion. Steps common to the cancer and non-cancer approaches were described above and will only briefly be reviewed, while details unique to the cancer methodology will be given a more thorough discussion.

Step 1: Identify Representative Chemicals

We selected from 1 to 10 cancer-causing chemicals for each problem area and identified the likely sources of these chemicals in Region I. The chemicals were selected based on our estimate of their contribution to the problem and the availability of data on exposures and health effects. At this point in the cancer analysis, three problem areas were categorized as not posing cancer risks. Criteria Air Pollutants (#1), Acid Deposition and Visibility (#2), and Lead (#22) were so characterized because of the existing presumption that the substances of concern are not currently considered to be carcinogens.

Step 2: Characterize Potency

The purpose of this step was to determine for each of the chemicals the relationship between the dose and the probability of developing cancer. This relationship was used to predict the level of risk associated with a given exposure. While there are various models for assessing carcinogenic potency, EPA has only approved the linearized multistage model. Using this model, it is assumed that at low doses the dose response curve is linear and that the slope goes through the origin (i.e., there is no threshold effects level for carcinogens). Therefore the simplified equation "Incremental Cancer Risk = Potency times Dose" can be used to determine an upper-bound probability of incremental cancer risk (i.e., cancers that would be expected above the background rate) for low doses of carcinogens. The work group adopted cancer potency factors derived from this model, where available. The potency factors were generated by EPA's Carcinogen Assessment Group (CAG).

Step 3: Assess Exposure

The purpose of this step was to estimate the concentration levels at which exposure occurs, identify the pathways and estimate doses, and estimate the size of the exposed population. The assessment was based on average, not worst-case, exposures and made use of available ambient monitoring or incidence data or modeling results. In most cases, exposures were assumed to be constant over a 70-year lifetime. At this point in the analysis, Accidental Releases (#17) were assumed to pose low cancer risks because chronic exposures were not a reasonable assumption. It is possible that a severe short-term exposure could cause cancer later in life, but we assumed that acute non-carcinogenic effects were of most concern for this problem area.

Step 4: Characterize Risk

In this step, exposure dose and potency estimates were multiplied together to calculate upper-bound individual excess cancer risks for each chemical. The individual risk was then multiplied by an estimate of the exposed population to give an estimate of the number of cancer cases expected over a period of 70 years. This number was then divided by 70 to provide an estimate of annual cancer incidence. The group agreed that it was reasonable to do this calculation to provide a relative estimate of annual cancer risk. For problem areas with multiple chemicals, the individual and population risks were each summed over the chemicals if it was assumed that individuals were exposed to the mixture.

Step 5: Estimate Percentage Covered and Uncertainty

Consistent with the overall methodology, qualitative estimates (i.e., high, medium, low) of the percentage of the problem covered (i.e., to what extent were we able to extrapolate to the Region as a whole from our analysis of a portion of the problem) and the uncertainty associated with the data were made for each problem area.

Step 6: Results of Analysis

A summary of the results of the cancer analysis is presented in Table II-1. The table displays the estimated individual risk, population risk (in cancers per year), the percentage of problem covered, and the estimated uncertainty.

For details on the data supporting the summary table, the reader is referred to the specific papers on the problem areas contained in the appendix. For some problem areas, no data appear because no quantitative analysis was performed. These problem areas were handled qualitatively by group consensus when it came time to perform the ranking. Problem areas #7, #8, and #9 are shown as a total risk from discharges to surface water. A qualitative attempt to differentiate between the three problem areas was deferred until the ranking.

Table II-1

Summary of Cancer Analysis

Problem Area	Individual Risk	Population Risk*	Percentage of Problem Covered	Uncertainty
1. Criteria Air Pollutants		NO CANCER RISK		
2. Acid Deposition and Visibility		NO CANCER RISK		
3. Hazardous/Toxic Air Pollutants	1E-4	~10	L	H
4. Radon	2E-4 to 4E-3	375-1500	H	L
5. Indoor Air Pollutants Other than Radon	0 to 1E-2	1-50	H	M
6. Radiation from Sources Other than Radon (non-ionizing)	NO QUANTITATIVE ANALYSIS PERFORMED			
7. Industrial Point Source Discharges to Surface Waters	1E-2	16-32	L	H
8. POTW Discharges to Surface Waters				
9. Nonpoint Source Discharges to Surface Waters				
10. Discharges to To Estuaries, Coastal Waters, and Oceans from All Sources	1E-4	90	M	H
12. Drinking Water	1E-4 to 1E-5	20	M	H
13. RCRA Waste Sites	1E-6	1-10	H	H
14. Superfund Waste Sites	1E-5	1	H	H
15. Municipal Waste Sites	1E-5	FEW	M	H
16. Industrial Waste Sites	1E-5	FEW	M	H
17. Accidental Releases		LOW CANCER RISK		
18. Releases from Storage Tanks	NO QUANTITATIVE ANALYSIS PERFORMED			
19. Other Ground-Water Contamination	1E-7	~1	L	H
20. Pesticide Residues on Food	2E-4	317	L	H
21. Pesticide Application		~1	L	H
22. Lead		NO CANCER RISK		
23. Asbestos	1E-4 to 1E-7	185	L	H

*Population risk expressed as excess cancers per year.

Note: L=low, M=medium, and H=high.

C. Non-Cancer Methodology

This section describes the non-cancer methodology in a step-by-step fashion. Steps common to the cancer and non-cancer approaches were described under the general approach and will only briefly be reviewed while details unique to the non-cancer methodology will be given a more thorough discussion.

Step 1: Identify Representative Chemicals

We selected from 1 to 10 non-cancer-causing chemicals for each problem area and identified the likely sources of these chemicals in Region I. Unlike the cancer analysis, each of the problem areas was characterized as posing some non-cancer risk.

Step 2: Determine Severity

The purpose of this step was to characterize the type and different severities of health endpoints of the selected chemicals. The work group considered a number of alternative schemes before adopting the severity index approach used in the NCRP. The NCRP approach will be outlined below, and the interested reader is referred to Appendix II of EPA's *Unfinished Business* for complete details.

In the NCRP approach, health effects were classified into 1 of 11 categories that were then broken down into 106 different health endpoints. A severity index basically ranked health effects by how threatening they were to the viability of the organism. This led to the development of a 7-point severity scale. The 106 separate endpoints were given scores from 1 to 7 based on the index. The 7-point index was later collapsed to a 4-point scoring scheme by combining severity scores 1 and 2 into one score, severity scores 3 and 4 into another score, and severity scores 5 and 6 into a third score. The 4-point scale better fit the NCRP's sense of the precision of the data with which they worked. Table II-2 shows the distribution of the endpoints on the 4-point scoring scale. It should be noted that this severity scoring approach has not been endorsed by EPA and is quite controversial. However, the NCRP recognized the need to make such rough judgments in order to analyze non-cancer risk. They did feel that there is a large difference in severity represented by a one point difference in the severity score.

The work group spent a significant amount of time discussing the pros and cons of this approach before adopting it. We decided that it best served the purposes of the work group and agreed that it would have taken a great deal of time and effort to improve the NCRP method. We also lacked sufficient expertise to devise a better alternative.

Table II-2

Distribution of Non-Cancer Health Endpoints on the Severity Scoring Scale

Score = 1

- herpes (non-infectious)
- liver-increased enzymes
- reduced corneal sensitivity
- sensory irritation
- giant cell formation
- nasal irritation
- nasal cellular irritation
- eye irritation
- dental mottling

Score = 2

- allergic reactions
- kidney-hyperplasia
- kidney-hypertrophy
- liver-increased weight
- decreased sensory perception
- irritability
- decreased testicular weight
- uterine hypoplasia
- pulmonary irritation
- nasal ulceration
- mucosal atrophy
- pulmonary congestion
- decreased mid-expiratory flow rates
- dental erosion
- leishmaniasis
- symptomatic effects (headache)
- increased blood pressure
- low birth weight
- decreased heme production
- impaired heme synthesis
- increased infections
- kidney atrophy
- jaundice
- multagenicity-cytogenic
- retinal disorders
- tremors
- post implanatation losses
- testicular degeneration
- spermato damage
- increased resorptions
- bronchitis
- pulmonary impairment
- lung injury

Score = 2
Continued

- respiratory-hemorrhage
- aggravation of asthma
- increased respiratory disease
- bronchoconstrictions
- increased respiratory infections
- gastrointestinal disease

Score = 3

- aggravation of angina
- bone marrow hypoplasia
- methemoglobinemia
- kidney-tubular degeneration
- hepatitis A
- AChE inhibition
- increased spontaneous abortion
- pneumonia
- Pontiac fever
- alveolar collapse
- fibrosis
- lung structure changes
- cataracts
- Legionnaires disease
- fetotoxicity
- kidney-necrosis
- liver-necrosis
- learning disabilities
- neuropathy
- convulsions
- aspermia
- emphysema
- pulmonary edema

Score = 4

- increased heart attacks
- teratogenicity
- mutagenicity-hereditary disorders
- retardation
- mortality

Note: 1=less severe; 4=most severe.

Step 3: Characterize Potency

The purpose of this step was to determine the relationship between the dose of a substance and the likelihood that the dose will produce an adverse health effect. Most non-carcinogenic dose-response functions are thought to involve a threshold below which it is unlikely that an adverse health effect will occur because, up to a certain dose, the human body is able to detoxify the chemical and repair any damage. The work group, after considerable discussion, decided again to adopt the NCRP approach. (The interested reader is referred to Appendix II of *Unfinished Business* for complete details.) The NCRP developed an "individual exposure ratio" that was calculated as the actual exposure dose divided by the Reference Dose or RfD. (The RfD is based on a no or lowest observed effects level in the study, divided by a safety factor normally ranging from 10 to 1,000.) The higher the exposure dose above the RfD, the higher the individual exposure ratio. At exposure doses below the RfD, we expect little likelihood of adverse effects occurring, while at levels above the RfD it is reasonable to assume that an adverse effect may occur in at least some sensitive population. This individual exposure ratio therefore provides some indication of the potency of these non-carcinogens relative to the estimated exposure dose. A four-point ratio score that reflects order of magnitude changes in the individual exposure ratio was then developed. This broad four-point scoring system once again reflected the NCRP's sense of the precision of the data available to them. The ratio scoring scheme is presented in Table II-3 (Section A).

For more than half of the problem areas, the exposure was judged to be below the RfD, producing an individual exposure ratio less than 1. In these cases no ratio score was assigned and it was assumed that there was negligible risk. This allowed us to keep separate those problems where there was a ratio in the 1 to 10 range from those where the ratio fell below 1.

For some problem areas, particularly in air programs, there are no established RfDs. In these instances the work group member investigating the problem developed a surrogate RfD after investigating other forms of existing standards, the values considered by the NCRP, and other suggestions from experts at EPA Headquarters.

Step 4: Assess Exposure

The purpose of this step was to estimate the likely concentration levels at which exposure occurs, the resulting doses, and the likely populations exposed. The assessment was based on average exposures and made use of the available data, whether measured or modeled. After much discussion, we adopted a modification of the NCRP population scoring approach for this assessment.

The NCRP approach converted the estimated population exposed to a chemical into a score from 1 to 4. One point of change in the score represents a difference in exposed population of two orders of magnitude. Thus errors of up to a factor of 10 in estimating exposed populations would make little difference in the final result. The work group modified the population ranges associated with each score to reflect populations more appropriate for the region. However, we maintained the NCRP's two orders of magnitude change in population between each score. Table II-3 (Section B) presents the work group's population scoring scheme.

Table II-3

Scoring System for Non-Cancer Risks

A. Ratio Score derived as follows:

Score	Individual Exposure Ratio
1	1 to 10
2	10 to 100
3	100 to 1,000
4	>1,000

B. Population Score derived as follows:

Score	Number of People Exposed
1	1 to 100
2	100 to 10,000
3	10,000 to 1,000,000
4	>1,000,000

C. Ratio Score derived from incidence data as follows:

Score	#Cases/#People Exposed	Individual Exposure Ratio
1	<1.00E-06	1 to 10
2	1.00E-06-1.00E-04	10 to 100
3	1.00E-04-1.00E-02	100 to 1000
4	>1.00E-02	>1000

In cases where incidence data were available, a different approach was used. The incidence data were converted to a population score and a ratio score to be consistent with the remainder of the non-cancer approach. A ratio score was calculated as the annual incidence divided by the estimated population at risk. Table II-3 (Section C) shows the ranges associated with each score and the individual exposure ratio for comparison. The population at risk was then converted into a population score.

Step 5: Characterize Risk

In this step the severity, potency, and exposure evaluations were combined to estimate non-cancer risk for each chemical and problem area. Since the work group had relied on a scoring system for the three components, we could not estimate individual and population risk in the same manner as we had in the cancer analysis. Instead, we had three separate scores for each chemical of concern in each problem area. We could combine the three scores in various ways to give us a relative sense of the differing risks posed by the problem areas.

In characterizing the population risk, the work group considered various ways of combining the severity, individual exposure ratio, and population scores. We finally decided to sum the three scores for each chemical analyzed in a problem area and assign the highest score within a problem area to that problem area. Other combination schemes, including averaging across chemicals within a problem area, made little or no difference in the relative scoring. A surrogate score for individual risk was developed by adding the severity and individual exposure ratio scores for each chemical and selecting the maximum sum within a problem area.

As mentioned in the potency discussion above, the estimated average exposure was below the RfD for about half the problem areas. Therefore, we did not assign an individual exposure ratio score. In characterizing the risk for these problem areas, we did not form a combined score but rather grouped these problem areas in a low risk category.

Step 6: Estimate Percentage Covered and Uncertainty

Consistent with the overall methodology, qualitative estimates of the percentage of the problem covered (i.e., to what extent were we able to extrapolate to the Region as a whole from our analysis of a portion of the problem) and the uncertainty associated with the data were made for each problem area.

Step 7: Results of Analysis

A summary of the results of the non-cancer analysis is presented in Table II-4. For each problem area the table displays the severity score, the individual exposure ratio or potency score, the population score, the percentage of problem covered, and the estimated uncertainty. For the problem areas where the individual exposure ratio was less than one, the individual and population risks are categorized as low rather than by a score. For details on the data supporting each of these scores, the reader is referred to the specific papers in the appendix on each problem area.

For some of the problem areas, no data appear because no quantitative analysis was performed. These problem areas were handled qualitatively by group consensus when the ranking was performed. As in the cancer analysis, problem areas #7, #8, and #9 are shown as a total risk from discharges to surface water. A qualitative attempt to differentiate between the three problem areas was postponed until the ranking was performed.

Table II-4

Summary of Non-Cancer Analysis

Problem Area	Severity Score	Potency Score	Population Score	Percentage of Problem Covered	Uncertainty
1. Criteria Air Pollutants	2	3	4	H	L
2. Acid Deposition and Visibility	3	3	4	H	H
3. Hazardous/Toxic Air Pollutants	NO QUANTITATIVE ANALYSIS PERFORMED				
4. Radon		LOW NON-CANCER RISK			
5. Indoor Air Pollutants Other than Radon	3	1	3	M	M
6. Radiation from Sources Other than Radon (non-ionizing)		LOW NON-CANCER RISK			
7. Industrial Point Source Discharges to Surface Waters					
8. POTW Discharges to Surface Waters	3	2	3	L	M
9. Nonpoint Source Discharges to Surface Waters					
10. Discharges to Estuaries, Coastal Waters, and Oceans from All Sources		LOW NON-CANCER RISK			
12. Drinking Water	2	3	3	M	H
13. RCRA Waste Sites		LOW NON-CANCER RISK			
14. Superfund Waste Sites		LOW NON-CANCER RISK			
15. Municipal Waste Sites		LOW NON-CANCER RISK			
16. Industrial Waste Sites		LOW NON-CANCER RISK			
17. Accidental Releases	3	2	2	M	M
18. Releases from Storage Tanks	3	NOT SCORED	3	L	H
19. Other Ground-Water Contamination	3	2	4	L	H
20. Pesticide Residues on Food		LOW NON-CANCER RISK			
21. Pesticide Application	2	2	3	L	H
22. Lead	3	4	3	H	L
23. Asbestos		LOW NON-CANCER RISK			

Note: L=low, M=medium, and H=high.

III. Relative Ranking

Once the risk analyses were completed, the work group met to perform a relative ranking of the problem areas for cancer and non-cancer risk. We considered options ranging from ordinal ranking to grouping into a few categories. We were not comfortable with developing an ordinal ranking because we did not believe our analysis provided us with anything approaching the level of precision needed to perform an ordinal ranking. We agreed to set a target of ranking the problems into five categories.

The ranking process was primarily influenced by the outcome of the two risk characterization efforts. It was supplemented by significant discussion and group consensus. For the problem areas where data gaps had prevented the calculation of an individual or population risk estimate, we worked with whatever data we had and our sense of the other problem areas to reach a consensus relative ranking.

Once the cancer and non-cancer rankings were completed, we embarked on the most difficult venture of all--developing an overall public health risk ranking. This chapter provides details on the process and the results of each of the three ranking exercises.

A. Cancer Risk

The work group's first cut at a relative ranking of cancer risk was based on the population risk which we had expressed as an annual cancer incidence. We had already created our lowest ranking group when we decided that Criteria Air Pollutants, Acid Deposition and Visibility, Accidental Releases, and Lead posed no or low cancer risks. Looking at the range of annual cancer incidence predicted in the analysis, the work group developed four additional ranges to use in the preliminary ranking. The worst (highest) ranking, Category 5, was defined as having an annual cancer incidence greater than 250 in the region. Category 4 was defined by the range of 100 to 250 annual cancers, Category 3 by 10 to 100 cancers, and Category 2 by 1 to 10 cancers. Category 1 contained the problem areas that posed no cancer risk.

The next step, for which we had very little data and high uncertainty, involved ranking Radiation from Sources Other than Radon and Releases from Storage Tanks. We elected to rank both in Category 2 based on a qualitative sense that there was some small risk. We also attempted to separate the risk from discharges to surface waters into the three separate problem areas. The risk estimates were driven by ingestion of fish contaminated with PCBs. The work group thought that Industrial Point Sources and Nonpoint Sources were the major contributors to this problem. POTWs were judged less likely to contribute to the risk, so we reduced the ranking for POTWs by one category. This determination was based on the types of contaminants contributing to the major risk.

At this point we reviewed the rankings to see if they made relative sense. Our review considered the individual risk, the percentage of problem covered, and the uncertainty. In this process, we revised the ranking of three problem areas. Hazardous/Toxic Air Pollutants, which was between Categories 2 and 3, was placed in Category 3 based on high individual risk and the low percentage of problem covered. Pesticide Residues on Food was dropped to Category 4 because of the work group's belief that the analysis scheme, based on NCRP data, had overstated the extent of the problem. Similarly, we thought the Asbestos analysis had overstated the problem and dropped it into Category 3.

The final results of the cancer ranking are presented in Table III-1. Within each category, problem areas are listed in the order they were originally listed for the project; no relative ranking within a category was performed or should be inferred.

Table III-1

Cancer Relative Risk Ranking
(unranked within categories)

High Risk	Category 5 Radon
	Category 4 Pesticide Residues on Food
	Category 3 Hazardous/Toxic Air Pollutants Indoor Air Pollutants Other than Radon Industrial Point Source Discharges to Surface Waters Nonpoint Source Discharges to Surface Waters Discharges to Estuaries, Coastal Waters, and Oceans from All Sources Drinking Water Asbestos
	Category 2 Radiation from Sources Other than Radon (non-ionizing) POTW Discharges to Surface Waters RCRA Waste Sites Superfund Waste Sites Municipal Waste Sites Industrial Waste Sites Releases from Storage Tanks Other Ground-Water Contamination Pesticide Application
	Category 1 Criteria Air Pollutants Acid Deposition and Visibility Accidental Releases
Low Risk	Lead

B. Non-Cancer Risk

The work group's first attempt at a relative ranking of non-cancer risk was based on the population risk estimate which we had expressed as a total score. The work group used the scoring range to develop five categories. Problem areas scoring 10 or more were put in Category 5, areas scoring 8 or 9 went in Category 4, areas scoring 6 or 7 went in Category 3, areas scoring 1 through 5 went in Category 2, and the problem areas that had been classified as "low" went in Category 1. The next step involved ranking Releases from Storage Tanks, for which we had little risk information. We elected to rank it in Category 3 based on a qualitative sense that the risks were on the same scale as other problem areas in this category. We also attempted to separate out the risks from discharges to surface waters. The risk estimates were driven by ingestion of fish contaminated with mercury and lead. The work group thought that Industrial Point Sources would likely be the major contributor, followed by Nonpoint Sources and then POTWs. POTWs and Nonpoint Sources were dropped down one category at this point.

The nature of the scoring scheme resulted in no problem areas listed for Category 2 and 12 problem areas listed in Category 1. The work group spent time seeing if it could subdivide this low-risk category into two groupings, but ultimately we could not. The non-cancer ranking thus shrunk to a four-category scale.

The work group then reviewed each of the problem areas, considering the individual risk, percentage of problem covered, uncertainty factors, and other information that might have come to light after the original analysis was conducted. This process resulted in revised scores for Criteria Air Pollutants (#1) and Acid Deposition and Visibility (#2). Criteria Air Pollutants had originally been ranked in Category 4, with a score of 9. This score was driven by increased asthmatic attacks and reduced activity in susceptible populations as a result of widespread violation of the Ozone National Ambient Air Quality Standard. Additional information indicates that levels at or near the standard result in respiratory effects in healthy adults and that repeated exposures heighten sensitivity. The summer of 1988 also showed an increase in days exceeding the standard over a wider area than in previous years. Upon consideration of this information, the work group moved Criteria Air Pollutants up to Category 5.

Acid Rain and Visibility had originally been ranked in Category 5, with a score of 10. This score was driven by increased hospital admissions due to respiratory illness resulting from inhalation of acidic aerosols, primarily sulfates. However, significant uncertainty surrounds the health effects data, which rely heavily on epidemiological studies. This uncertainty led the work group to lower the relative ranking to Category 4. Health effects from total particulate exposures (including the acid aerosols) would also support a ranking in Category 4.

The final results of the non-cancer ranking are presented in Table III-2. Within each category, problem areas are listed in the order they were originally listed for the project; no relative ranking within a category was performed or should be inferred.

Table III-2

Non-Cancer Relative Risk Ranking
(unranked within categories)

High	Category 5
Risk	Criteria Air Pollutants
	Lead
	Category 4
	Acid Deposition and Visibility
	Industrial Point Source Discharges to Surface Waters
	Drinking Water
	Other Ground-Water Contamination
	Category 3
	Indoor Air Pollutants Other than Radon
	Nonpoint Source Discharges to Surface Waters
	Accidental Releases
	Releases from Storage Tanks
	Pesticide Application
	Category 2/1
	Hazardous/Toxic Air Pollutants
	Radon
	Radiation from Sources Other than Radon (non-ionizing)
	POTW Discharges to Surface Waters
	Discharges to Estuaries, Coastal Waters, and Oceans from All Sources
	RCRA Waste Sites
	Superfund Waste Sites
	Municipal Waste Sites
	Industrial Waste Sites
Low	Pesticide Residues on Food
Risk	Asbestos

C. Overall Risk

The final work group task was clearly the most difficult one: developing an overall relative public health risk ranking. The work group spent more than two meetings on this task and discussed many ways to approach the problem. We could not develop a clear-cut method for combining the cancer and non-cancer risks. Instead, our ranking grew from a group consensus-building process and ultimately represents our best collective professional judgment.

One path we examined involved using explicit approaches to combine the cancer and non-cancer rankings. These approaches included averaging the rankings, using various weighted averages, or using the highest of the cancer or non-cancer rankings to characterize a problem area. All of these approaches are fraught with the difficulty of comparing very different health effects. It is ultimately a personal value judgment whether a lung cancer case later in life is better, equivalent, or worse compared with a lifetime with learning disabilities because of childhood lead poisoning. Not surprisingly, the work group members' judgments in evaluating these types of questions differed greatly.

The other major path we examined involved attempting to suspend judgment on the thorny questions posed above, focusing instead on those problem areas that were likely to be causing the worst public health problems in the region. This did not mean turning our backs on the results of our earlier analysis. Instead, it meant considering to what extent the problem area posed risks beyond levels that might be broadly portrayed as acceptable. This approach relies on the fact that, as regulators, we have set standards for many substances, both carcinogens and non-carcinogens, and have decided that exposures above these levels are unacceptable. We could then consider the level and extent of these unacceptable exposures in grading the problem areas. Using this approach, we would not be explicitly equating cancer and non-cancer effects but rather would be saying that both can be bad above certain exposure levels.

The work group ended up using a combined approach. Each member prepared his or her own individual ranking, dividing the problem areas into four or five groups. Each person was free to devise his or her own approach to combining the cancer and non-cancer rankings. Many people used some explicit scheme as a starting point and then made adjustments based on their professional judgments. Other rankings were based primarily on well-informed judgments but were not explicitly driven by the cancer and non-cancer rankings. As one might have expected, all of the individual rankings were different.

We then analyzed the individual work group member's rankings and identified the problem areas that fell into the same ranking category in the majority of the rankings. About two-thirds of the problem areas were thus sorted into five ranking categories. (The remaining problem areas had split rankings, falling between the categories sorted out above.) We then reviewed the relative ranking of the problem areas that had been placed in the same categories in the majority of the individual rankings. We discussed each problem area, reviewing the data we had analyzed and its uncertainty and comparing it with other problem areas in the same ranking category. After these discussions, none of the initial problem area rankings was changed. This process also provided a good benchmark for ranking the remaining areas.

For the remaining problem areas, we used a similar process of reviewing the available information for each problem area and comparing the risk to the risks of problem areas already ranked. During the discussion we considered creating a sixth ranking category. We rejected this idea because we did not believe the data available allowed us to make that many gradations with confidence.

The most difficult part of the process was agreeing on the highest risk category. We had used Radon, which had received the highest cancer risk rating and also showed some small non-cancer risk, as the benchmark for the highest risk category. We discussed at length whether Criteria Air Pollutants and Lead, which had received the highest non-cancer risk rating but low/no cancer risk, should join Radon in the highest overall risk category. We ultimately did rank them in the same category. We recognize that the health effects posed by each of these problem areas are very different and difficult to equate. However, we can comfortably state that these three problem areas pose the most significant residual public health risks in the region and that they stand apart from the other problem areas in this respect.

Once we determined the high-risk category, the remaining problem areas fell fairly easily into place. The final ranking reflects the work group's consensus. While some individuals might have preferred to see a particular problem area shifted by one category, as a work group we reached a point where no person was uncomfortable with the final result. We agreed that the uncertainty in the process made it difficult to draw very hard lines between adjacent ranking categories, especially in the middle of the range. At the same time we are confident that none of the problem areas were off by two ranking groups.

The final relative ranking of residual public health risks posed by the problem areas is displayed in detail in Table III-3. The problem areas have been placed into five categories of decreasing magnitude. Within each category, the problem areas are listed in the order in which they were originally listed for this project. No relative ranking within a category was performed and none should be inferred. Within the table, the first column lists the problem areas and the second column indicates the chemicals analyzed. The third column summarizes our findings in both the cancer and non-cancer analyses and other relevant comments. A brief summary, showing only the groupings of the problem area titles into the five categories, is displayed in Table III-4.

Some of the major conclusions of the Public Health Risk Work Group include the following:

- Indoor radon exposure, which is associated with lung cancer, poses a significant residual health risk in Region I. The estimated excess annual cancer cases associated with radon exposure may be up to an order of magnitude greater than those for other problem areas that pose a cancer risk.

- Lead exposure from all sources poses a significant public health threat in New England, particularly to young children who may suffer from learning disabilities as a result of mild to severe lead poisoning.

- Exposure to ground-level ozone concentrations above the National Ambient Air Quality Standards occurs frequently in the summer in Region I and is associated with respiratory ailments that may affect large populations in New England.

- Pesticide residues on food are a significant public health concern in Region I. The analysis available to us simply scaled a national estimate down to a regional estimate, based on a straight ratio of regional to national population. The absence of regional data made us reluctant to place it in the highest category. Nevertheless, pesticide residues on food consumed in New England are a significant concern.

- Separating the residual public health risk posed by the three types of discharges to surface waters was difficult. After much discussion and input from Water Management Division staff, we agreed to rank these problems as follows: industrial point sources in Category 4, nonpoint sources in Category 3, and POTWs in Category 2. This determination was based on the types of contaminants contributing to the major risk.

- Most of the waste-related problems, including RCRA, Superfund, municipal, and industrial waste sites, pose low residual public health risks. These problem areas primarily show up as ground-water contamination problems that may or, more likely, may not be affecting drinking water supplies today. While these sources are contaminating aquifers that may be valuable future sources of drinking water, current exposures involve relatively low concentrations and relatively small populations.

- The work group agreed that, although our risk analyses would indicate that municipal landfills pose risks similar to Superfund sites, there is great uncertainty related to municipal landfills and the problem would greatly benefit from a more in-depth analysis.

Table III-3

Relative Residual Public Health Risk Ranking
(unranked within categories)

Problem Area	Substances/Exposures Investigated	Comments
Category 5		
Criteria Air Pollutants	Ozone Carbon Monoxide Particulate Matter	Cancer: Category 1–Assumed no cancer risk since all criteria air pollutants are currently considered non-carcinogenic Non-cancer: Category 5–Driven by large-scale exposure to high ozone levels across the region
Radon	Radon	Cancer: Category 5–Up to 1,500 excess cancers predicted annually from exposure in homes Non-cancer: Category 2/1–Assumed low; some non-cancer effects likely
Lead	Lead	Cancer: Category 1–Assumed no cancer risk since chemical is not currently considered carcinogenic Non-cancer: Category 5–Driven by ingestion of soil or inhalation of lead-contaminated dust by children, resulting in lead poisoning and learning disabilities
Category 4		
Acid Deposition and Visibility	Acid Aerosols–Sulfates and Nitrates	Cancer: Category 1–Assumed no cancer risk since substances are not currently considered carcinogenic Non-cancer: Category 4–Influenced by widespread respiratory symptoms and hospital admissions due to inhalation of elevated acid aerosol levels
Indoor Air Pollutants Other than Radon	Carbon Monoxide Nitrogen Oxides Particulate Matter Formaldehyde Benzene Carbon Tetrachloride Phthalate Esters Chlordane Heptachlor Tobacco Smoke Trichloroethylene Tetrachloroethylene	Cancer: Category 3–Estimated 1 to 50 excess annual cancers from inhalation of a mixture of pollutants; high individual risk Non-cancer: Category 3–Driven by carbon monoxide exposures, aggravating angina for chronic effects and causing acute cardiac impairment
Industrial Point Source Discharges to Surface Waters	PCBs, Dioxin, Mercury	Cancer: Category 3–Estimated 15 to 30 excess annual cancers from ingestion of fish contaminated with PCBs Non-cancer: Category 4–Influenced by ingestion of mercury contaminated fish at up to 25 times the RfD
Drinking Water	Total Trihalomethanes (TTHM), Arsenic, Radionuclides, Nitrates, Bacteria, Pathogens	Cancer: Category 3–Estimated 20 excess annual cancers from ingestion of TTHM and arsenic Non-cancer: Category 4–Driven by outbreaks of Giardia-related gastrointestinal disease

(continued)

Table III-3 (Continued)

Relative Residual Public Health Risk Ranking
(unranked within categories)

Problem Area	Substances/Exposures Investigated	Comments
Category 4 (continued)		
Other Ground-Water Contamination	Pathogenic Microorganisms, Sodium, Nitrates, Pesticides, Solvents, and Petroleum Products	Cancer: Category 2–Estimated 1 excess annual cancer from ingestion of dichloromethane from septic tanks/cesspools Non-cancer: Category 4–Driven by outbreaks of hepatitis and gastrointestinal disease caused by septic tank contamination of drinking-water wells
Pesticide Residues on Food	1 Herbicide 2 Fungicides 1 Insecticide 1 Growth Regulator	Cancer: Category 4–Estimated up to 320 excess annual cancers by scaling NCRP data down to region, a method that may overestimate the regional impact Non-cancer: Category 2/1–Assumed low non-cancer risk because of lack of evidence that exposures exceed allowable levels
Category 3		
Hazardous/Toxic Air Pollutants	TCE Carbon Tetrachloride	Cancer: Category 3–Estimated 10 excess annual cancers based on monitored background concentrations of urban toxic soup; may underestimate effect because of low percentage of problem covered Non-cancer: Category 2/1–Quantitative risk not estimated because of lack of inhalation reference doses to evaluate systemic effects from these pollutants
Asbestos	Asbestos	Cancer: Category 3–Estimated 185 excess annual cancers; work group felt that the estimate was conservative Non-cancer: Category 2/1–Assumed low non-cancer risk
Discharges to Estuaries, Coastal Waters, and Oceans from All Sources	Heptachlor PCBs DDT HCB	Cancer: Category 3–Estimated 90 excess annual cancers from ingestion of contaminated fish Non-cancer: Category 2/1–Estimated low risk since exposures do not exceed RfDs
Nonpoint Source Discharges to Surface Waters	PCBs Dioxin Mercury	Cancer: Category 3–Estimated less than 30 excess annual cancers from ingestion of fish contaminated by PCBs; not judged as significant a contributor as industrial point sources Non-cancer: Category 2/1–Qualitatively estimated as smaller contributor to surface waters than industrial point sources

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(continued)

Table III-3 (Continued)

Relative Residual Public Health Risk Ranking
(unranked within categories)

Problem Area	Substances/Exposures Investigated	Comments
Category 2		
POTW Discharges to Surface Waters	PCBs Dioxin Mercury	Cancer: Category 2–Estimated less than 10 excess annual cancers from ingestion of fish contaminated with PCBs; judged a smaller contributor than nonpoint or industrial point sources Non-cancer: Category 2/1–Qualitatively estimated as a smaller contributor to surface waters than nonpoint or industrial point sources
Accidental Releases	Chlorine Mineral Acids Ammonia Organic Solvents Organic Toxics	Cancer: Category 1–Assumed low cancer risk Non-cancer: Category 3–Influenced by respiratory problems as severe as respiratory pneumonia; estimates based on reported accident data
Releases from Storage Tanks	Gasoline Home Heating Oil Diesel Fuel	Cancer: Category 2–Qualitative ranking based on best judgment of risk relative to problem areas with more data; judged similar to hazardous waste risks Non-cancer: Category 3–Qualitative ranking based on best judgment of risks relative to other problem areas; high number of tanks drives higher rating
Pesticide Application	Herbicides Fungicides Insecticides	Cancer: Category 2–Estimated less than 1 excess annual cancer; derived by scaling down national figures Non-cancer: Category 3–Qualitative ranking based on best judgment of risks relative to other problem areas
Category 1		
Radiation from Sources Other than Radon (non-ionizing)	Extremely Low Frequency (ELF) Radiation	Cancer: Category 2–Qualitative ranking based on best judgment of risks relative to other problem areas; high degree of uncertainty in available data Non-cancer: Category 2/1–Qualitative ranking based on best judgment of risks relative to other problem areas

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(continued)

Table III-3 (Continued)

Relative Residual Public Health Risk Ranking
(unranked within categories)

Problem Area	Substances/Exposures Investigated	Comments
Category 1 (continued)		
RCRA Waste Sites	18 Typical Compounds in Waste Stream	Cancer: Category 2–Estimated 1 to 10 excess annual cancers, influenced most heavily by modeled hazardous waste incineration impacts; high degree of uncertainty Non-cancer: Category 2/1–Estimated low risk since exposures do not exceed RfDs
Superfund Waste Sites	PCBs, Arsenic, Tetrachloroethylene, Benzene, Vinyl Chloride	Cancer: Category 2–Estimated about one excess annual cancer Non-cancer: Category 2/1–Estimated low risk since exposures generally do not exceed RfDs
Municipal Waste Sites	Vinyl Chloride Arsenic Tetrachloroethylene Dichloromethane Carbon Tetrachloride	Cancer: Category 2–Qualitatively estimated a few excess annual cancers due to contamination of ground-water wells; limited available data Non-cancer: Category 2/1–Estimated low risk since exposures do not exceed RfDs
Industrial Waste Sites	Vinyl Chloride Arsenic Tetrachloroethylene Dichloromethane Carbon Tetrachloride	Cancer: Category 2–Qualitatively estimated a few excess annual cancers due to contamination of wells; limited available data Non-cancer: Category 2/1–Estimated low risk since exposures do not exceed RfDs

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Table III-4

Summary of Relative Residual Public Health Risk Ranking (unranked within categories)

High Risk	**Category 5**
	Criteria Air Pollutants
	Radon
	Lead
	Category 4
	Acid Deposition and Visibility
	Indoor Air Pollutants Other than Radon
	Industrial Point Source Discharges to Surface Waters
	Drinking Water
	Other Ground-Water Contamination
	Pesticide Residues on Foods
	Category 3
	Hazardous/Toxic Air Pollutants
	Nonpoint Source Discharges to Surface Waters
	Discharges to Estuaries, Coastal Waters, and Oceans from All Sources
	Asbestos
	Category 2
	POTW Discharges to Surface Waters
	Accidental Releases
	Releases from Storage Tanks
	Pesticide Application
	Category 1
	Radiation from Sources Other than Radon (non-ionizing)
	RCRA Waste Sites
	Superfund Waste Sites
	Municipal Waste Sites
Low Risk	Industrial Waste Sites

IV. Observations

In the nine months from the Public Health Risk Work Group's first meeting to the final ranking session, we learned much about the relative residual public health risk in the region and perhaps even more about risk assessment and group decision making. This section presents the most important of the work group's observations after reflecting on where we have been and what we have learned. Many observations are not surprising, but they all offer valuable lessons for those who might venture down a similar path in the future.

- **Good data are hard to come by.**

The work group ended up analyzing far fewer data than we had originally envisioned. We attributed this to two major problems. First, data we thought or hoped existed simply did not exist or existed in such limited quantities that they were of little use. In the second instance, the data existed in some form but were so decentralized and stored in so many different ways that it would have taken significant time and money to compile them. State agencies possess vast quantities of valuable data that are often extremely inaccessible. We need to put more resources into developing and improving data management in Region I and in the states.

- **Lack of a standard non-cancer methodology made this analysis difficult.**

The varied approaches to regulating non-carcinogens across the agency make it very difficult to employ a consistent analytical methodology. Working with LOELs, NOELs, ADIs, RfDs, and varying safety factors quickly leads to a methodological headache. It is also difficult to determine how to measure severity of effect. While we would not portray the cancer analysis as simple, it looks easy by comparison. If the agency continues to advocate the use of comparative risk analyses, it would be well served to build a consistent non-cancer approach.

- **Residual risk differs from total risk.**

We evaluated residual risk, that is, the risk posed by current exposures, given the current level of control. In a number of problem areas, the risks we estimated were smaller than we thought at the outset. In many cases this is due to some level of existing success in addressing the total risk posed by the problem area. Once existing controls are factored in, a significant percentage of the risk posed by a problem area may be reduced. High residual risk is often indicative of the absence of a regulatory program or a lack of success in existing programs. Similarly, some problem areas have lower residual risk because the regulatory programs have been successful. One cannot readily determine from our analysis how the relative risk would change if existing regulatory programs were de-emphasized.

- **There are some real differences in risk among areas.**

Despite data limitations, methodological headaches, and the reliance on professional judgment, the work group is reasonably confident that there are different categories of public health risk in Region I.

- **It is difficult to combine cancer and non-cancer risks.**

An enormous amount of personal value judgment is involved in trying to compare and sum cancer and non-cancer health risks. Although none of the work group members thought it would be easy, few were prepared for just how uncomfortable and difficult the process proved to be. Our consensus judgment was well informed and carefully nurtured, but it cannot be validated or replicated.

Appendix

Problem Area Papers

1. Criteria Air Pollutants

Problem Area Definition

This category covers exposure to criteria air pollutants regulated under the Clean Air Act for the protection of human health and public welfare. The human health effects were evaluated for the three criteria air pollutants (ozone, particulate matter less than 10 microns[1] and carbon monoxide) that exceed the National Ambient Air Quality Standards (NAAQS) in Region I. Other criteria air pollutants such as oxides of nitrogen and sulfur were not included in this evaluation because New England is currently meeting the NAAQS for those substances, and we simplified our analysis by assuming that exposures below the NAAQS posed no risk. Lead, the remaining criteria air pollutant, was evaluated as a separate problem area. Major sources of the pollutants evaluated include motor vehicles, industries, and fuel burning operations.

Summary/Abstract

This assessment focused on the non-carcinogenic health effects attributed to ozone (O_3), particulate matter less than 10 microns (PM_{10}), and carbon monoxide as a result of exposures to levels in excess of the NAAQS. Carcinogenic effects were not considered in this assessment because cancer is not thought to be of concern from exposure to these pollutants. The health effects of concern that served as the basis for the NAAQS and also for our analysis include increased risk of asthmatic attacks (O_3), restricted activity (O_3 and PM_{10}), aggravation of angina (CO), and even premature death (PM_{10}). These effects represent significant health effects and, as such, the severity scores assigned to these pollutants were some of the highest of any problem area evaluated. The uncertainty in our evaluation was low, owing to well-documented health effects and a reliable monitoring network from which reasonable exposure information was obtained. Because the focus of the analysis was on health effects in excess of the NAAQS and we could identify the non-compliance areas within the region, we felt that a large portion of this problem was being addressed. Health effects associated with levels below the NAAQS were not considered.

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[1] Subsequent to this analysis, the Public Health Risk Work Group decided that there was substantial overlap between the particulate matter health effects analysed here and the effects of acid aerosols--one portion of the particulate matter dose--analyzed under Acid Deposition and Visibility (problem area #2). We decided to combine the particulate matter and acid aerosol health risks to eliminate this overlap. We combined them under Acid Deposition and Visibility since the acid aerosol analysis indicated the greater public health risk. Therefore, the particular matter analysis described in this paper was actually considered as part of the Acid Deposition and Visibility problem area in ranking the public health risk.

Toxicity Assessment

By definition, we were concerned with only those criteria air pollutants for which New England currently exceeds the NAAQS. Consequently, ozone, PM_{10}, and carbon monoxide were our pollutants of concern. Other criteria air pollutants (oxides of sulfur and nitrogen, and lead) were not evaluated because Region I is currently meeting the NAAQS.

Because each of the three pollutants evaluated are not currently considered to be carcinogenic, we focused our evaluation on the systemic effects of these substances. The health effects of concern are those which served as the basis for EPA's National Ambient Air Quality Standards. As such, the health effects of concern are as follows: for ozone--asthma attacks and respiratory restricted activity days; for particulate matter--premature death and restricted activity; and for carbon monoxide--aggravation of angina. The National Ambient Air Quality Standards we focused on are 0.12 ppm (one hour average) for O_3, 50 ug/m^3 (annual average) for PM_{10}, and 9 ppm (eight-hour average) for carbon monoxide.

Exposure Assessment

Sources of carbon monoxide and PM_{10} include automobiles, diesel engines, and combustion sources. Ozone is not emitted directly into our atmosphere but is created by the interaction of sunlight with hydrocarbons and NO_x. Thus, many of the large industrial facilities that emit volatile organic compounds, combustion sources, cars, and trucks emit precursors of ozone.

The only pathway of exposure considered for this problem area was through direct inhalation.

Information from the 1986 Annual Air Quality Report for New England was used to obtain data on the number, location, and actual pollutant levels in excess of the NAAQS from monitors located throughout New England. These data were used with probabilistic equations developed by a consultant for Region X to estimate the actual incidence of different health effects attributable to ozone and PM_{10}. Because a probabilistic equation was not available for carbon monoxide, we made a crude estimate of the number of people exposed to levels in excess of the eight-hour standard and attempted to characterize the population at increased risk of angina.

The population at risk of experiencing an asthma attack as a result of exposure to ozone levels in excess of the standard are those who experience levels in excess of the standard and who are asthmatics. Because asthma attacks may be brought on by acute exposures to ozone occurring repeatedly throughout the course of a year, we built in a measure of exposure frequency into our population estimate. Thus, for ozone, the population at risk translates to approximately 2.8 million person-days based upon estimates of the population exposed to ozone levels in excess of the standard (72 million person-days) and the prevalence of asthmatics in the population (approximately 4 percent). The remaining 70 million person-days in Region I were evaluated as being at risk of experiencing restricted activity on days when ozone levels exceeded the standard.

For particulate matter, only one monitoring location in all of Region I exceeded the annual average. Thus the population in this city (New Haven, CT--population 120,000) was deemed at increased risk of premature death and restricted activity.

For carbon monoxide, eight urban areas with a total population of 1.2 million people were deemed at risk of exposure to carbon monoxide levels in excess of the eight-hour standard. Of these people, approximately 96,000 were deemed to be at increased risk of having their angina aggravated as a result of exposure to carbon monoxide based on the prevalence of angina (8 percent) in the U.S. population.

Risk Characterization

Cancer risk estimates were not performed because the pollutants evaluated are not thought to be known or suspect human carcinogens.

The general approach we used to assess the non-cancer health effects of criteria air pollutants was to generate estimates of annual incidence. To do this we relied on several equations developed by a contractor working on this problem for another region. The equations predicted incidence given exposure information, the population, and pollutant levels. This was done for both ozone and PM_{10} because we had the incidence equations developed for Region X's Comparative Risk Project. For carbon monoxide, an incidence equation was not available; thus a simple ratio of the exposure levels to the eight-hour standard was used to describe potency. The results of our calculations follow:

- **Ozone**

For ozone, two health endpoints were evaluated: increased asthmatic attacks and restricted activity days. These two endpoints were both given a moderate severity ranking (2 on a scale of 1 to 4). The incidence values computed using Region X's equation together with Region I exposure information resulted in annual incidence estimates of approximately 3.7E-4 and 1.2E-3 for asthmatic attacks and for restricted activity days, respectively. This incidence information (substitute for potency estimate) justified issuing a high potency score to each of these endpoints (3 on a scale of 1 to 4). Because the populations for both endpoints were very large (in excess of one million), the highest population score (4) was assigned to each.

- **Particulate Matter**

Premature deaths were estimated based on exceedances of the annual average standard and a probability (incidence) equation developed by a contractor for Region X. Possible premature deaths were considered extremely severe and thus warranted the highest severity of effect ranking score. The predicted incidence of death given Region I exposure information was 5E-6, which translated to a mild potency value of 2. The population at risk was assumed to be the entire urban population (120,000) of the city in which the annual average was exceeded, which placed this endpoint at the high end (population score of 3). Restricted activity (severity score 2) was also evaluated. The predicted incidence was much higher (4E-2), warranting a potency score of 4. The population at risk was the same population as above, namely the 120,000 urban dwellers, corresponding to a population score of 3.

- **Carbon Monoxide**

Since incidence data were not readily available and since we did not have any means to estimate annual incidence, the average CO levels in the CO non-attainment areas were compared to the NAAQS to compute a potency score. Since the levels in exceedance areas were only slightly above the eight-hour standard, the potency score assigned to CO was low (1). The health effect of concern at the standard is aggravation of angina, which warranted a severity score of 3. The population estimates were based on the number of people who have heart conditions in areas currently exceeding the NAAQS. This was estimated to be approximately 8 percent of the urban population from several different urban areas, or approximately 96,000 individuals, warranting a population score of 3.

Because cancer effects were not evaluated for this problem area, only the non-cancer effects had to be combined. To accomplish this, each of the severity, potency, and population scores were summed for each pollutant evaluated.

Uncertainty

The uncertainty in this analysis is attributed predominantly to the exposure analysis; the hazard identification of acute effects has been studied extensively and is well documented. We only considered effects associated with exposure to levels above the current NAAQS. Thus, we may have overlooked health effects associated with exposure to levels below the standard. The 1986 Ambient Air Quality Report for New England served as our source of data on documented exceedances. Although we can be very confident of the monitoring data, it is limited to a few locations in each state. As a result, the areal extent of actual violations of the NAAQS may be underrepresented. For these reasons, the risks associated with this problem area may be underestimated.

Estimates of the number of people in the vicinity of each monitor (NO_X, CO) were crude at best, and for ozone we assumed that entire urban populations were exposed. Estimates of the exposure to ozone in excess of the standard, reported in terms of person-days, were generated by staff in the Air Management Division.

Because incidence estimates were generated using an equation which did not receive much scrutiny from the Regional Office, the incidence estimates for the health effects associated with ozone and PM_{10} are subject to some uncertainty. However, the uncertainty assigned to this problem area was deemed to be low in comparison to the other problem areas.

We felt that the percentage of problem covered in this analysis was high even though the focus was only on three pollutants. This is because Region I is currently in attainment for SO_x, NO_x, and lead.

2. Acid Deposition and Visibility

Problem Area Definition

This problem area concerns health effects associated with acidic aerosols and acid deposition. Some gases emitted into the atmosphere react with sunlight, water vapor, and oxygen to form acid compounds. When these compounds are suspended in the atmosphere, they are referred to as acidic aerosols.[1] Acid deposition occurs when these compounds interact with precipitation or with small particles to deposit on land and water surfaces.

The primary precursor pollutants that lead to acidic aerosol formation are sulfur dioxide and nitrogen dioxide. Sources of these primary pollutants include electric utilities (especially coal-fired), industrial boilers and heaters, smelters, residential and commercial fuel burning, and mobile sources. Emissions of volatile organic compounds also contribute to the formation of acidic aerosols.

Most of the acidic aerosols that form in the atmosphere consist of very fine acid sulfate and nitrate particles. The health effects of primary concern here are associated with the inhalation of sulfate aerosols. Other possible effects, associated with the leaching of toxic metals into drinking water, will be evaluated in the drinking water problem area.

Summary/Abstract

The risks associated with this problem area include respiratory symptoms, hospital admissions, and possibly elevated mortality rates due to the inhalation of acidic aerosols. The population exposed is large (more than 1 million people), but the population at risk for some endpoints of concern is much smaller. The incidence-to-population ratios are fairly high (about 3E-2 for respiratory symptoms and 2E-5 for death). Hospital admissions due to respiratory illness were calculated to pose the highest risk for the endpoints analysed, with a potency score of 3, a population score of 4, and a severity score of 3. (The maximum score for any of the three factors was 4.) The percentage of problem covered was considered high, and the uncertainty was considered high as well.

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[1] Subsequent to this analysis, the Public Health Risk Work Group decided that there was substantial overlap between the effects of particulate matter analyzed under Criteria Air Pollutants (problem area #1) and the effects of acid aerosols--one portion of the particulate matter dose--analyzed here. We decided to combine particulate matter and acid aerosol health risks to eliminate this overlap. We combined them under Acid Deposition and Visibility because the acid aerosol analysis indicated the greater public health risk. Therefore, the particulate matter analysis described in the Criteria Air Pollutants paper was actually considered as part of the Acid Deposition and Visibility problem area in ranking the public health risk.

Toxicity Assessment

Evidence suggests that exposures to acidic aerosols are associated with respiratory symptoms, such as cough, in adults and children with chronic respiratory diseases as well as in healthy individuals. Associations have been observed between sulfate levels and mortality rates and hospital admissions due to respiratory illnesses. Laboratory work with animals suggests that acidic aerosols could be related to the development of chronic respiratory diseases, but this has not been confirmed with human epidemiological studies.

We have estimated four categories of health effects (deaths, hospital admissions, respiratory symptoms in children, and respiratory symptoms in adults) using the following equations recommended by our expert consultant, RCG/Hagler, Bailly, Inc.:

Annual deaths = .000037 * (Sj - 10) * POPj

where: Sj = annual average sulfate level in area j (ug/cubic meter)
POPj = population in area j

Annual admissions = .000086 * Sj/9 * POPj

Children with respiratory symptoms = .035 * C

where: C = the number of children in areas where the annual average sulfate level exceeds 10 ug/cubic meter

Adults with respiratory symptoms = .05 * M + .02 * W

where: M = the number of men in areas where the annual average sulfate level exceeds 10 ug/cubic meter
W = the number of women in areas where the annual average sulfate level exceeds 10 ug/cubic meter

The work group assigned the following severity scores to the health endpoints of concern for the non-cancer health effects analysis:

deaths: 4
hospital admissions: 3
respiratory symptoms: 1

Exposure Assessment

For the exposure assessment, we used the available monitoring data on ambient sulfate levels in the region as an indicator of the population exposure to potentially harmful acidic aerosols. Some data points were eliminated from further consideration in order to exclude a few local source-dominated impacts. The group felt it was not reasonable to associate these hot-spot impacts with larger populations that are probably exposed to lower levels of sulfates. In addition, some older data were eliminated because the group felt they were no longer representative of current ambient levels.

In most cases where a city had more than one sulfate monitor, monitor readings were averaged to obtain a single characteristic value for the city. Sulfate levels in a city were associated with the city population. Rural monitor levels within a county were averaged, and the resulting average concentration was associated with the county population. If the county included cities for which monitoring data were available, then the rural monitor levels were associated only with the rural population in the county. Sulfate levels were not estimated for counties without monitors.

The number of individuals in New England exposed to acidic aerosols is greater than one million, which translates to a population score of 4 in our ranking system. However, the number of individuals at risk for some of the endpoints of concern (those exposed to annual average levels greater than 10 ug/cubic meter for death and respiratory symptoms) is much lower.

Risk Characterization

A small number of excess annual deaths was predicted using the methodology suggested by the consultant. The analysis for deaths produced an estimated incidence/population ratio of 2E-5 for the population at risk. This corresponds to a potency score of 2 in our scoring system, with a population score of 3 and a severity score of 4.

The analysis for increased hospital admissions due to respiratory illnesses produced an incidence/population ratio of 1.2E-5 for the population at risk. This represents a potency score of 3, with a population score of 4 and a severity score of 3.

The methodology recommended by the consultant for estimating the increased incidence of respiratory symptoms in children implicitly uses an incidence/population ratio of 3.5E-5 for the population at risk. The analysis for the increased incidence of respiratory symptoms in adults produced an incidence/population ratio of approximately 2.6E-6. These represent potency scores of 4, with a population score of 3 and a severity score of 1.

The analytic procedures used to estimate the incidence of health effects are based on epidemiological study results that demonstrate associations, but not causation. Therefore, there is some uncertainty regarding the magnitude and possibly the existence of the assumed causal relationships used here to relate pollutant levels to human health effects.

The equations recommended by the consultant to estimate excess mortality and increased respiratory symptoms make use of a threshold value of 10 ug/cubic meter. The selection of this specific threshold value is somewhat arbitrary, since the available scientific information does not allow for a more precise estimation of a suitable threshold.

The results of the exposure and risk analyses are sensitive to how multiple data points in cities are treated and to what populations are selected to associate with monitored levels. Very few recent monitor sites exhibited annual sulfate levels greater than 10 ug/cubic meter. One site in Maine was not carried through the full analysis because the work group believed that the monitored levels represented elevated impacts from a local source and were not indicative of the type of problem for which this problem area was created. The remaining monitor site with recent annual sulfate levels greater than 10 ug/cubic meter was in Springfield, Mass. Another site in Springfield exhibited annual levels somewhat lower. The work group decided to associate the higher concentration measurement with the population of Springfield, rather than averaging the levels at both monitors. Averaging the two monitored levels would have produced an average concentration below 10 ug/cubic meter and eliminated the excess mortality and increased respiratory health effects entirely.

In these analyses, we relied upon the use of available information on ambient sulfate levels as an indicator of population exposures to potentially harmful acidic aerosols. It must be noted that certain types of sulfates, such as sulfuric acid, may be more harmful than other types of sulfates and that acidic nitrates may also represent a potential health hazard. The proportion of the total problem represented by sulfates would be expected to vary, and therefore it is difficult to specify what portion of the harmful acidic aerosol total is represented by sulfates. Our consultant thought that sulfates represented much more of the problem than nitrates.

Another factor to consider is the correlation between sulfate levels and levels of total particulates. Attributing the health effects of concern here entirely to acidic aerosols may somewhat overestimate the degree of their culpability.

The uncertainties associated with the use of ambient sulfate levels as an indicator and with the correlation between sulfate and total particulate levels may be reduced when more monitoring data for a variety of acidic aerosols become available, and when more epidemiological studies are able to make use of such data.

An important potential health effect that was not considered in the analysis is the development of chronic respiratory disease. Although it may be reflected to some extent in the estimates of other health effects that were considered, chronic respiratory disease may represent another important health problem associated with the long-term exposure to elevated acidic aerosol levels.

The combined effect of these uncertainties is not clear. To the extent that some health effects due to other pollutants are being attributed to acidic aerosols, these estimates may be too high. Conversely, to the extent that significant health effects are not considered and to the extent that other acidic aerosols are not accounted for in the analysis, these estimates may be too low.

The work group considered the percentage of the problem covered to be high and the uncertainty to be high as well.

3. Hazardous/Toxic Air Pollutants

Problem Area Definition

This problem area evaluates risks from ambient exposure to hazardous air pollutants. This is defined to include a very broad set of substances that currently are not directly regulated under the Clean Air Act, although they may indirectly be controlled as components of regulated mixtures such as volatile organic compounds. Monitoring data for chromium, arsenic, benzene, perchloroethylene, trichloroethylene, and carbon tetrachloride were considered. Because monitoring data are so limited and poorly organized, modeling data were also evaluated for a broader range of pollutants.

Sources of these pollutants include point sources such as incinerators, fugitive emissions from solvent use, and area sources such as motor vehicle emissions.

Summary/Abstract

Individual risks based on monitoring data and professional judgment were estimated to be in the 1E-4 range for lifetime exposure. The population exposed to this risk was estimated to be 3.4 million. Data provided by a modeling analysis predicted higher risks. Risk estimates were not considered from "high risk point sources" but only from background urban exposures. Non-cancer risk estimates were also not considered because of the lack of inhalation Reference Doses to evaluate systemic effects from these pollutants.

The data used to make these risk estimates have a large range of uncertainty due to the lack of routine monitoring for these pollutants, the lack of standard methods for such monitoring, and an inability to predict how well ambient air data serve as an estimate of actual human exposure.

Toxicity Assessment

Because of the broad range of pollutants considered, it is not possible to present detailed discussions of the toxicity of these pollutants. Attachment 3-A shows unit risk estimates for the six pollutants evaluated based on monitoring data. These include two metals and four VOCs. The metals have unit risk estimates $[ug/m^3]^{-1}$ in the 1E-2 to 1E-3 range, while the VOCs have unit risk estimates in the 1E-6 range.

Hazard assessments for these chemicals are based on data of widely varying quality and certainty. Arsenic, chromium, and benzene are considered by EPA to be class A carcinogens, indicating that there are human data available to support the conclusion that they are carcinogens. There is a major uncertainty about chromium, however, since it is considered that only Cr+6 and not Cr+3 is carcinogenic.

Data on many VOCs are based on animal bioassays. There is considerable controversy about many of these VOCs, such as perchloroethylene, over what their hazard classification should be. Perchloroethylene, in particular, is now somewhere between a "C" (possible human carcinogen) and a "B2" (probable human carcinogen) in the agency's ranking. These uncertainties, which tend to get lost in quantitative evaluations of risk, need to be considered. In summary, a small number of these pollutants are almost certainly human carcinogens while there is great uncertainty about the rest.

There is even greater uncertainty concerning non-carcinogenic or systemic effects of these pollutants. Other than the criteria pollutants such as lead or particulate matter, the air program has not evaluated systemic effects for air pollutants in any systematic way. This is in contrast to EPA's evaluation of oral exposure where "risk reference doses" (RfDs) are well established for a large number of pollutants. There is a work group developing inhalation RfDs within the agency, but its methods are not yet final.

Other approaches, such as use of occupational standards divided by safety factors, have also been used, particularly by state air programs. These were not considered useful for this analysis, however. As a consequence, only cancer risks are considered, using standard CAG unit risk estimates. These estimates assume lifetime exposure at a breathing rate of 20 cubic meters per day.

Exposure Assessment

The most difficult aspect of this analysis was the compilation and evaluation of exposure data. This difficulty can be attributed to the lack of a regulatory structure to control these pollutants. Because there is no federal regulatory program, sources of monitoring data are limited to a small number of research programs and special studies. In addition, the data that are available have not been compiled in a systematic way that would allow a comprehensive evaluation of risks. Finally, even where data are available, there are major uncertainties caused by the lack of standard air toxics monitoring methods.

There were, however, some data that were evaluated from the following sources:

- EPA's Toxics Air Monitoring System (TAMS): Chelsea, MA; Boston, MA (VOCs)
- EPA's NMOC Program: Portland, ME; Boston, MA; New Haven, CT; and Bridgeport, CT (VOCs)
- EPA's National Particulate Filter Analysis Program: 25 cities (metals)
- A report from the MA air toxics program (VOCs)

Data from the first three of these sources were entered into a Lotus spreadsheet. The Massachusetts study was reviewed and compared with data from the other sources. The standard averaging time for these studies was 24 hours. Although several days of observations were available in most cases, extrapolating from a small number of 24-hour observations to annual levels introduces another level of uncertainty.

The metals data showed large variations in concentrations for arsenic, cadmium, nickel, and chromium. Of these four, the data for arsenic and chromium appeared to be the most consistent and were used to develop the risk estimates discussed later. A summary of this data is presented in Table 3-1, along with risk estimates.

A major risk issue with chromium concerns the percentage that is Cr+6 as opposed to Cr+3, because only Cr+6 is considered to be carcinogenic. For the risk estimates developed here, it was assumed that one-seventh of the total Cr was Cr+6. This ratio had been used in an epidemiological study of Cr health effects, but could have a large margin of error.

The VOC data were much more limited in geographic scope, but for the TAMS and NMOC studies analyses had been done for dozens of organic compounds. Four VOCs were used to develop risk estimates: benzene, perchloroethylene, trichloroethylene, and carbon tetrachloride. These were chosen for their prevalence and for the availability of unit risk estimates. A number of other VOCs, such as 1,1,1-trichloroethane, 1,1-dichloroethene, and various aromatics, were omitted. The data are summarized in Table 3-1.

No ambient data were found for formaldehyde, which in other studies has been shown to contribute to a significant fraction of the overall risk from ambient exposures.

It should be noted that all of these data pertain to background urban exposures ("urban soup"). Sources of such exposures include motor vehicle emissions, combustion of fuel in boilers, solvent use by dry cleaners and other commercial establishments, and emissions from gasoline stations.

It does not address levels in non-urban areas (which are likely to be lower), and it also does not address exposure from "high risk point sources" around which levels of these and other pollutants are likely to be much higher. Such high risk sources include chemical production plants, combustion units such as waste incinerators, and processors of large amounts of solvents.

In addition to this monitoring data, modeling data were developed by a consultant (but completed after the work group ranking). These data were developed from national emission estimates for a large number of pollutants. The advantage of these data are that they are much more comprehensive than the monitoring data in geographic scope and in number of pollutants covered. The disadvantage is that the emission estimates on which the modeling is based have a large potential margin of error. In addition, uncertainty is introduced because of the modeling.

Modeled data were available for 22 pollutants. A summary of this data is provided in Table 3-2. As can be seen from comparing Tables 3-1 and 3-2, the modeling data predict much higher risks than the available monitoring data. This in part is due to more conservative assumptions used in the modeling (such as 24 hours per day exposure versus 8 hours per day assumed for the evaluation of the monitoring data) but may also be due to the tendency of the emission estimates to overstate actual emissions. At this point, the only statement that can be made is that there is a discrepancy which warrants further evaluation.

It is likely that the monitoring data set an upper bound on the risk levels from ambient exposures to hazardous air pollutants, not considering the exposure to "high risk point sources".

Risk Characterization

Risk has been characterized only for the carcinogenic risks of hazardous air pollutants. As noted above, standard methods are not available for developing inhalation risk reference doses, so non-cancer effects have not been considered. There would also be some potential double-counting with the criteria pollutant evaluation. It should be realized, however, that in addition to the carcinogenic effects discussed below, these pollutants could also be present in amounts large enough to cause concern about systemic effects.

Table 3-1 summarizes the risk evaluation based on monitoring data. Using mean values of these data, individual risks for each pollutant are generally in the 1E-6 range, while cumulative risks are in the 1E-5 range. The evaluation assumes eight hours per day exposure, and assumes that Cr+6 is one-seventh of total Cr. The evaluation does not include a large number of pollutants for which ambient data were not available. It was, therefore, the consensus of the work group that the actual risk of the total mixture of pollutants (including such substances as formaldehyde and BAP) was likely to be in the 1E-4 range.

The population to which this risk applies is considered to be those living in urban areas--about 3.4 million, based on 1980 census data. In addition, there are transient populations, such as commuters, which are exposed to these pollutants for a shorter length of time. Using the 3.4 million population figure, lifetime risks are in the range of 70 (for the 6 pollutants considered) to 700 (assuming a 10-fold increase in risk when other pollutants are added).

As noted above, the modeling data predict higher lifetime risks. As shown in Table 3-2, best-case lifetime population risk for all pollutants is about 1,800 cancer cases, while worst-case estimates are about 8,300. The worst-case estimate is about an order of magnitude higher than for the monitoring data risk assessment while the best-case estimate is about a two-fold increase over the monitoring estimates.

Table 3-1

Summary of Air Toxics Monitoring Data

Pollutant	Number of Sites	(UG/L)			Unit Risk $(ug/m3)^{-1}$	Individual Risk
		Maximum	**Minimum**	**Mean**		
Chromium(+6)	25	0.0659	0.0000	0.0049	1.02E-02	2.38E-06
Arsenic	25	0.0110	0.0000	0.0024	4.30E-03	3.44E-06
Benzene	2	3.0200	0.0000	1.6700	8.03E-06	4.47E-06
Perchloroethylene	2	295.7000	0.0000	16.4900	5.80E-07	3.19E-06
Trichloroethylene	1	1.9600	1.0500	1.4900	1.30E-06	6.46E-07
Carbon Tetrachloride	1	1.7800	0.8900	1.3400	1.50E-05	6.70E-06

Total	Urban Population	Total Risk
2.08E-05	3.40E+06	70.80

Notes: Assumes Cr+6=1/7 of total Cr.
Assumes exposure = 8/24 hours/day.

Table 3-2

Summary of Air Toxics Modeling Data
Lifetime Population Cancer Risk

Pollutant	Best Case	Worst Case
Arsenic	8.3E+01	4.0E+02
BAP	1.1E+01	7.0E+01
Benzene	1.7E+02	9.7E+02
Beryllium	1.5E+00	8.0E+00
1,3 Butadiene	3.6E+00	2.9E+02
Formaldehyde	3.9E+02	1.7E+03
EDB	4.8E+01	2.2E+02
Cadmium	1.2E+01	6.7E+01
Chloroform	9.8E+00	5.2E+01
Carbon Tetrachloride	2.7E+01	1.1E+02
Gasoline Vapors	1.1E+00	8.9E+01
Cr+6	8.8E+02	3.5E+03
Methylene Cl	9.0E+01	5.1E+02
PCB	5.8E+00	2.3E+01
PERC	6.9E+00	5.7E+01
POM	1.4E+00	3.9E+01
TCE	4.5E+00	4.7E+01
Vinylidene Cl	6.1E+01	2.4E+02
	1,806	8,392

4. Radon

Problem Area Definition

This problem area covers risks from exposure to indoor radon through all routes of exposure, including inhalation of indoor air and exposure to radon gas released from drinking water. Only indoor exposures are considered.

Summary/Abstract

Radon is a naturally occurring inert radioactive gas. Radon 222 is produced as a decay product of Uranium 238, which is found naturally in certain rock types, and has a half life of 3.8 days. Radon decays into other short-lived radioactive progeny which can be inhaled and adsorbed within the lungs. Sources of radon include rocks and soil around a home and underground water from uranium-bearing aquifers, although migration of radon through soil pore space and into a home is considered to be the major source of indoor exposure.

An increased risk of developing lung cancer is the health effect of concern resulting from exposure to elevated levels of radon. Nationally, EPA estimates that 5,000 to 20,000 lung cancer deaths per year can be attributed to radon. These risk estimates are based on epidemiological studies of miners, and employ use of a "relative risk" model that ties the risk of lung cancer due to radon to the overall rate of lung cancer in the country. Risks of lung cancer from combined exposure to radon and smoking are greater than the sum of risks from exposure to either one alone.

Region I has maintained a database of indoor radon measurements that was used to develop regional risk estimates for this study. These estimates were based on more than 4,000 basement measurements taken on both a random and non-random basis in all Region I states. Using the risk equation that the Office of Radiation Programs (ORP) developed to estimate national risks, it was estimated that 375 to 1,500 lung cancer deaths per year could be attributed to radon exposure. An additional analysis of annual cancer incidence attributable to ingestion and inhalation of radon from drinking water was conducted (see page 20). It is estimated that approximately 75 additional annual cases could be attributed to this route of exposure, for a total range of 450 to 1,575 cancers per year.

Toxicity Assessment

As stated above, risks from radon are due to exposure to decay products. Collectively, these decay products are measured in units of working levels (WL), although radon itself is usually measured in picocuries per liter (pCi/L). In order to develop risk estimates based on radon concentration data, it is necessary to convert from pCi/L to WL, and, for this analysis, a standard conversion factor of 0.005 is used.

The equation ORP used to develop national radon risk estimates is shown in Attachment 4-A. Elements of this equation are discussed briefly here. A more detailed discussion can be found in EPA's *Radon Reference Manual*, EPA 520/1-87-20. The first element (C_R) is the average indoor radon decay product concentration that, for the national estimate, was one average for the whole country. As discussed below, the regional estimate was based on a range of radon measurements broken into percentiles.

The second element (T) is the time (in hours) of exposure to radon which is assumed. The ORP equation assumes lifetime exposure, but also assumes 75 percent occupancy in the home and subtracts a 10-year latency period. The third factor (F_{WLM}) converts WL hours to WL months, which is the unit for which risks to miners has been developed. This factor also takes into account the difference in breathing rates between a miner and the average adult.

The next factor (R_{RRM}) is the estimate of relative risk per WL month. This factor is based on the relative risk model which assumes that the risk of developing a tumor because of radon exposure is relative to the underlying lung cancer rate in the overall population. The relative risk model projects an expected percentage increase in lung cancer rates. The percentage is based on epidemiological studies of lung cancer rates in miners. About 13 such studies have been conducted. There is a range of relative risks that can be developed from these studies, and the range that ORP uses (which has been supported by the Science Advisory Board) is 1 percent to 4 percent.

The next factor is the underlying annual average lung cancer rate (TCR), which (from 1980 vital statistics) is 4.584E-4 per person. The last factor (POP) is the population to which the risk estimates are applied. For the national estimate, the total U.S. population was used. For the regional analysis, an estimate of the population living in detached structures was used.

Exposure Assessment

Low levels of radon can be found everywhere. In the indoor environment, exposure to radon mainly results from radon in rocks and soil containing uranium or radium. Radon gas can then enter a home through floors or walls as a result of either diffusion or pressure driven flow. Radon can also enter a home through ground water that contains radon as a result of the rock formation from which it comes. The radon is then released when water is used in the home.

The regional estimate of radon risk was developed based on data taken from more than 4,000 New England homes. These data come from a variety of sources including random studies of homes in CT and RI and non-random data supplied by radon testing companies in the other states. All of the data used were from tests done in basements where radon levels are expected to be highest. These tests are done using charcoal canisters which serve as a short-term screening method to detect radon levels during a 24- to 48-hour period.

Attachment 4-B summarizes these data. For this analysis, the measurements were broken into more than 80 fractions based on the range of radon concentration measured. For each fraction, the percentage of total measurements in that fraction was determined. (For example, about 22 percent of homes had radon levels between 1 and 2 pCi/L). The measurements in pCi/L were then converted to WL.

The next step was to determine the population in each fraction. To do this, 1980 census data were used. Since the measurements and the associated risk apply to persons living in single family homes or in other one-story dwellings (such as duplexes), an estimate of that population was made by taking the number of single family homes (2.49 million) and multiplying it by the average number of persons per household (2.74) to get an exposed population of about 6.8 million.

An estimate of persons living on the ground floor of multi-unit buildings was added to this number by taking the number of persons living in households with 2 to 4 units (about 3 million) and assuming that 50 percent of persons living in those households lived on the ground floor (about 1.5 million). Adding this to the previous figure gives a total estimate of about 8.3 million for the population at risk.

It was then assumed that this population was exposed to the same distribution of radon concentrations as was the sample in the regional data base. The population was then distributed according to the distribution of measurements in that data base. Basement measurements were converted to an estimate of radon levels in living areas by multiplying by a correction factor of 0.3. The ORP radon risk equation was then used to estimate a low (assuming a 1 percent relative risk) and a high (assuming a 4 percent relative risk) risk estimate.

Risk Characterization

A shown in Attachment 4-B, the evaluation estimates between 375 and 1,500 annual lung cancer deaths that can be attributed to radon. This corresponds to an annual individual risk for the exposed population of about 4.5E-5 to 1.8E-4.

The analysis has a number of strengths and uncertainties. First, the estimates of radon risk are based on human data, in contrast to many environmental pollutants for which toxicity is evaluated from data derived from animal studies. In addition, the range of radon concentrations found in homes is not, in many cases, different from the concentrations found in the mines where the studies were done. Therefore, there is less of a problem extrapolating from high to low doses. Finally, the exposure data are based on a relatively large number of samples taken within homes. This provides the basis for some confidence that the concentrations evaluated in this risk evaluation are representative of levels to which people are actually exposed.

There are also many uncertainties in the evaluation. The relative risk model assumes that risks from radon are tied into the overall lung cancer rate. Most of this lung cancer can be attributed to smoking, and there is uncertainty as to how much of the risks from radon exposure are due to combined radon/smoking effects. ORP has attempted to estimate what percentages of the total lung cancer deaths are due to smoking, radon, and smoking/radon. These data show that if smoking were eliminated the number of cases caused in part by radon would decrease greatly.

There are other uncertainties in the risk estimates, such as the quality of the miner data and the extrapolation from studies of healthy male workers to the general population. It should be noted that the National Academy of Sciences recently evaluated radon risks and came up with estimates that were close to ORP's.

There is also uncertainty in the exposure estimates due to the extrapolation from basement to living area concentrations, in the estimate of the population exposed, and in the assumption that persons are exposed throughout their lifetimes to the radon levels that were evaluated.

The percentage of problem covered is high. However, it should be noted that radon exposure through ground water was not explicitly evaluated. The radon data base may have missed this route of exposure since basement measurements are designed to capture radon exposure through rock and soil emissions and not through household water use.

Radon in Drinking Water

Summary

Individual cancer risk derived from exposure to radon in drinking water is about 8.3E-4. The annual cancer incidence attributable to ingestion and inhalation of radon from drinking water is approximately 75 cases.

Hazard Assessment

Radon is a natural occurring radionuclide and is a human carcinogen (Group A). The Cancer Potency Factor (CPF, or slope factor) is 2E-8 to 6E-6 per pCi/L. A CPF of 5E-7 per pCi/L of radon in water was used in this analysis.

Exposure Assessment

The radon data were based on the following: *Radon in Vermont Public Water Supplies* (1986), *A Survey of Radon-222 Occurrence in Connecticut Private Well Water; Assessing Geologic and Hydrologic Parameters* (1987), and the monitoring data compiled by the Water Supply and Ground Water Management Branch in Region I.

The median concentration of radon in drinking water was estimated to be 1,745 pCi/L. The exposed population was estimated to be 6 million ground-water users in Region I.

Routes of exposure considered in this evaluation were ingestion and inhalation of the radon emitted into the air as a result of all household water supply uses. Exposure from dermal contact is considered minimal.

Risk Characterization

The individual cancer risk is estimated at 8.3E-4. The annual cancer incidence attributable to radon exposure from drinking water is approximately 75 cases.

Uncertainties

Radon data from various sources are used. The validity of the data and their representation are difficult to evaluate. The CPF estimated for radon is 2E-8 to 6E-6 per pCi/L of radon in water. A CPF of 5E-7 per pCi/L for radon is chosen to estimate the cancer risk derived from exposure to this compound from drinking water. It is uncertain whether the selection of 5E-7 per pCi/L is an underestimate or an overestimate.

Attachment 4-A

Simplified Approximation of Estimates For Total U.S. Lung Cancer Deaths Due to Indoor Radon

A. Equation

Total U.S. Lung Cancer Death From Indoor Radon (1980) $= C_R * T * F_{WLM} * R_{RRM} * TCR * POP$

where:

C_R = average (mean) lifetime indoor radon decay product concentration = 0.004 WL-life

T = average interval of lifetime exposure in hours, following a 10-year minimum induction period during which no lung cancer will be observed, assuming 75% occupancy and 73.88 years life span (1980 vital statistics) $= 0.75 * (73.88-10) * 365 * 24 = 419{,}691.6$ hours/life

F_{WLM} = factor converting average cumulative indoor exposure in WL hours to working level months (WLM) for a miner (since risk estimates are based on miner data), accounting for 170 hours per month exposure period per WLM (by definition), and the difference in breathing rate between the average adult (15.3 liters per minute) and a miner (30 liters per minute) $= 1/170 * 15.3/30 = 0.003$ WLM per hour

R_{RRM} = relative lung cancer risk for lifetime exposure to radon, per WLM, using relative risk model = 1% to 4% per WLM

TCR = underlying annual average of U.S. lifetime lung cancer risk (1980 vital statistics) $= 4.584 * 10^{-4}$ per person

POP = 1980 U.S. population =226,545,805

B. Calculation

$$\begin{aligned}\text{Total Lung Cancer Deaths} &= 0.004 * 419{,}691.6 * 0.003\\ &\quad * 0.01 \text{ (lower risk estimate)}\\ &\quad * 4.584 * 10^{-4} * 226{,}545{,}805\\ &= \underline{5{,}230}\end{aligned}$$

$$\begin{aligned}\text{Total Lung Cancer Deaths} &= 0.004 * 419{,}691.6 * 0.003\\ &\quad * 0.04 \text{ (upper risk estimate)}\\ &\quad * 4.584 * 10^{-4} * 226{,}545{,}805\\ &= \underline{20{,}921}\end{aligned}$$

C. Notes

The above calculations differ from the estimates in "A Citizens Guide" of 5,000 to 20,000 lung cancer deaths principally due to two simplifications in the equation used above:

- The factor F_{WLM} does not include correction for the smaller lung size and lower breathing rate of children. Both are, in fact, recognized in the detailed analysis.

- The product of TCR and POP is replaced in the detailed analysis by calculations using 1980 mortality rates and 1980 life table statistics. The detailed actuarial analysis more properly accounts for latency effects, competing risks, and the lower underlying risk of lung cancer at young ages and, hence, results in a lower estimate.

Use of the 10-year latent period leaves an average life span of 63.88 years (73.88 years-10 years) during which the potential excess lung cancer risk can be expressed.

Source: Putnam, Hayes & Bartlett, Inc., September 1987. The numerical assumptions listed in Part A of this table are based on EPA analysis. Specific sources are noted in the text.

Approximate Yearly Lung Cancer Deaths from Exposure to Airborne Radon in New England

pCi/L Range	Fraction in Range	WL Range	Population in Range	Lower Risk Estimate	Upper Risk Estimate	Overall Risk Estimate # Lung Cancer Deaths/Year	
						Lower	Upper
1	0.287394	0.005	2402042	20.80	83.18	376	1505
2	0.223230	0.01	1865764	32.31	129.22		
3	0.141530	0.015	1182914	30.72	122.89		
4	0.080049	0.02	669054	23.17	92.68		
5	0.060037	0.025	501790	21.72	86.88		
6	0.037961	0.03	317283	16.48	65.92		
7	0.026820	0.035	224167	13.58	54.34		
8	0.020012	0.04	167263	11.58	46.34		
9	0.017949	0.045	150020	11.69	46.76		
10	0.016711	0.05	139674	12.09	48.37		
11	0.008665	0.055	72423	6.90	27.59		
12	0.008046	0.06	67250	6.99	27.95		
13	0.008665	0.065	72423	8.15	32.60		
14	0.004332	0.07	36212	4.39	17.56		
15	0.007839	0.075	65526	8.51	34.04		
16	0.002063	0.08	17244	2.39	9.55		
17	0.005364	0.085	44834	6.60	26.39		
18	0.003713	0.09	31039	4.84	19.35		
19	0.002269	0.095	18968	3.12	12.48		
20	0.002475	0.1	20692	3.58	14.33		
21	0.002682	0.105	22417	4.08	16.30		
22	0.002475	0.11	20692	3.94	15.76		
23	0.001444	0.115	12071	2.40	9.61		
24	0.001237	0.12	10346	2.15	8.60		
25	0.001237	0.125	10346	2.24	8.96		
26	0.001650	0.13	13795	3.11	12.42		
27	0.001650	0.135	13795	3.22	12.90		
28	0.001856	0.14	15519	3.76	15.05		
29	0.001031	0.145	8622	2.16	8.66		
30	0.001031	0.15	8622	2.24	8.96		
31	0.000618	0.155	5173	1.39	5.55		
32	0.000412	0.16	3449	0.96	3.82		
33	0.000206	0.165	1724	0.49	1.97		
34	0.000825	0.17	6897	2.03	8.12		
35	0.000412	0.175	3449	1.04	4.18		
36	0.001031	0.18	8622	2.69	10.75		
37	0.000825	0.185	6897	2.21	8.84		
38	0.000206	0.19	1724	0.57	2.27		

(continued)

Approximate Yearly Lung Cancer Deaths from Exposure to Airborne Radon in New England (continued)

pCI/L Range	Fraction in Range	WL Range	Population in Range	Lower Risk Estimate	Upper Risk Estimate	Overall Risk Estimate # Lung Cancer Deaths/Year	
						Lower	Upper
39	0.000825	0.195	6897	2.33	9.32		
40	0.000618	0.2	5173	1.79	7.17		
41	0.000412	0.205	3449	1.22	4.90		
42	0.000618	0.21	5173	1.88	7.52		
43	0.000412	0.215	3449	1.28	5.14		
44	0.000412	0.22	3449	1.31	5.25		
45	0.000618	0.225	5173	2.02	8.06		
46	0.000206	0.23	1724	0.69	2.75		
48	0.000412	0.24	3449	1.43	5.73		
50	0.000618	0.25	5173	2.24	8.96		
52	0.001031	0.26	8622	3.88	15.53		
53	0.000412	0.265	3449	1.58	6.33		
54	0.000206	0.27	1724	0.81	3.22		
55	0.000206	0.275	1724	0.82	3.28		
56	0.000206	0.28	1724	0.84	3.34		
58	0.000206	0.29	1724	0.87	3.46		
59	0.000412	0.295	3449	1.76	7.05		
60	0.000206	0.3	1724	0.90	3.58		
61	0.000206	0.305	1724	0.91	3.64		
62	0.000412	0.31	3449	1.85	7.40		
64	0.000206	0.32	1724	0.96	3.82		
66	0.000206	0.33	1724	0.99	3.94		
72	0.000412	0.36	3449	2.15	8.60		
76	0.000206	0.38	1724	1.13	4.54		
81	0.000206	0.405	1724	1.21	4.84		
84	0.000206	0.42	1724	1.25	5.02		
87	0.000206	0.435	1724	1.30	5.20		
90	0.000206	0.45	1724	1.34	5.37		
98	0.000206	0.49	1724	1.46	5.85		
100	0.000206	0.5	1724	1.49	5.97		
106	0.000206	0.53	1724	1.58	6.33		
109	0.000206	0.545	1724	1.63	6.51		
115	0.000206	0.575	1724	1.72	6.87		
116	0.000206	0.58	1724	1.73	6.93		
122	0.000206	0.61	1724	1.82	7.29		
124	0.000206	0.62	1724	1.85	7.40		
130	0.000206	0.65	1724	1.94	7.76		
135	0.000206	0.675	1724	2.02	8.06		
158	0.000206	0.79	1724	2.36	9.43		
187	0.000206	0.935	1724	2.79	11.17		
207	0.000206	1.035	1724	3.09	12.36		
243	0.000206	1.215	1724	3.63	14.51		
326	0.000206	1.63	1724	4.87	19.47		
335	0.000206	1.675	1724	5.00	20.00		
417	0.000206	2.085	1724	6.23	24.90		

(continued)

Approximate Yearly Lung Cancer Deaths from Exposure to Airborne Radon in New England *(continued)*

EPA equation: Lung Cancer Deaths = CR * T * FWLM * RRRM * TCR * POP

WL/pCi/L= 0.005 Conversion from pCi/L to Working Level
(assumes 50% equilibrium).

Population:
Single = 6847000
Duplex = 1511000 Note:Actual 2-4 family units = 3022000.
Use 1/2 to estimate side-by-side duplexes.

Const = 0.57716 = T * FWLM * TCR

RRRMlow = 0.01
RRRMhi = 0.04

Liv/Bas = 0.3 Assumes the lived in area is, on average
only 30 % of the basement concentration.

Note: Uses the calculation in the EPA Radon Reference manual.
Uses data on radon measurements in basements for the six New England states and EPA/Region I.

5. Indoor Air Pollutants Other than Radon

Problem Area Definition

This category includes exposures to air pollutants in schools, offices, homes, and indoor spaces from sources in the indoor environment. Common sources of indoor air pollution include cleaning supplies, paints, gas stoves, pressed wood products, upholstery, pesticides, and cigarettes. Indoor air pollutants include environmental tobacco smoke, formaldehyde, benzene, carbon monoxide, carbon tetrachloride, trichloroethylene, oxides of nitrogen, chlordane, heptachlor, and phthalate esters.

Summary/Abstract

A draft EPA report, *Indoor Air Pollution, The Magnitude and Anatomy of Problems and Solutions: A Scoping Study* (1987), served as the resource for much of the exposure information. Information presented in this report, which summarized monitoring information and reporting time weighted exposure levels by building type, was relied on quite heavily. This information was combined with Region I specific population statistics, obtained from the census bureau, in order to evaluate both carcinogenic and non-carcinogenic (acute and chronic) health effects. The computed upper-bound estimate of the individual lifetime cancer risk from exposure to indoor air pollutants was approximately 1E-2 and the population risk for Region I was approximately 1E-3 (annual incidence of 50). The chronic non-cancer health effects were projected for a large population but were of relatively low potency (exposure levels were only slightly greater than the standard).

The computed cancer and non-cancer health effects assume simultaneous exposure to each of the nine carcinogens and three non-carcinogens evaluated, which is most likely a conservative assumption. Additionally, the number of individuals in New England who actually have sources of pollution in their indoor environments and those at risk of experiencing adverse health effects were extremely difficult to estimate and may not be an accurate representation. The cancer potency factors and the health effects associated with the non-carcinogens have been studied quite extensively. In general, we are confident about the hazard identification portion but less certain about the exposure assessment. The percentage of problem covered was high due to the relatively large number of chemicals evaluated.

Toxicity Assessment

Selecting just a handful of chemicals for evaluation from the many hundreds of indoor air pollutants we are exposed to every day was a somewhat difficult task. We attempted to select chemicals on the basis of their toxic effects, the availability of exposure information, their prevalence in indoor environments, and their ability to serve as surrogates of exposure for other, similar chemicals.

The known or suspected human carcinogens selected for evaluation include the following volatile organic chemicals: benzene, carbon tetrachloride, tetrachloroethylene, trichloroethylene, and formaldehyde. Pesticides evaluated include chlordane and heptachlor. Environmental tobacco smoke and phthalate esters were also evaluated for carcinogenic effects.

Substances evaluated for their non-carcinogenic effects include carbon monoxide, oxides of nitrogen, respirable particulates, and formaldehyde.

Health Effects of Concern

Non-Carcinogenic

The National Ambient Air Quality Standards (NAAQS) for CO, NO_x, and respirable particulates served as the standard for acute and chronic exposures. A recommendation put forth by the American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE) for formaldehyde was used to assess the potential for health effects due to this substance.

The non-carcinogenic endpoints that formed the basis for our standard-setting process (NAAQS) were selected as the health effects of concern for this problem area. The health effects of concern associated with chronic and acute exposures to indoor air pollutants include cardiac impairment (CO); increased respiratory illness and decreased lung function (NO_x); impaired lung defense mechanisms, increased lung resistance, and a worsening of pre-existing lung disease (respirable particulates); and eye and respiratory irritation (formaldehyde). Health effects that may occur at levels of exposure below the acute/chronic standards were not evaluated.

Cancer Potency

Preference was given to using potency factors for the inhalation route of exposure, but in the absence of a potency factor based on an inhalation study, oral potency factors were used. The potency factor for environmental tobacco smoke was not assessed. Instead, cancer risk estimates were based on incidence data generated by leading researchers in this field. Potency factors expressed in (mg/kg/day)-1 are as follows:

benzene	0.029
carbon tetrachloride	0.13
tetrachloroethylene	0.0017
trichloroethylene	0.0046
formaldehyde*	0.038
phthalate esters**	0.00067
chlordane	1.3
heptachlor	4.5

* Based on a value reported in the EPA 1987 Assessment of Health Risks to Garment Workers and Home Residents from Formaldehyde.

** Based on unit risk for di-(ethylhexyl)-phthalate.

Exposure Assessment

Common sources of indoor air pollutants include cleaning supplies (solvents), paints, gas stoves, combustion sources, pressed wood products, upholstery, urea formaldehyde foam insulation, pesticides, and cigarettes. These sources are in our homes, offices, schools, and public buildings.

For this category, we considered direct inhalation exposures resulting primarily from the volatilization or off-gassing of pollutants generated from activities that take place in indoor environments (i.e., burning wood or a cigarette produces particulates, formaldehyde, NO_x, CO, and benzene).

As mentioned above, a draft EPA report on indoor air pollution served as the basis for much of the exposure information. Assumptions used in the exposure assessment were heavily based on the assumptions from the draft EPA report. As a result, three common indoor exposure environments were selected as the foci for this problem area: homes, offices, and schools. Because people readily move about, and the pollutants and the exposure concentrations differ from location to location, we took into consideration human activity patterns when estimating average daily exposures. In order to do this, we assumed that we all breathe, on average, 1.3 m^3 of air each hour and that over the course of a lifetime we spend:

- 16 hrs/day at home every day
- 6 hrs/day at school, 180 days/yr for 12.6 years
- 7.5 hrs/day at the office, 250 days/yr for 45 years

Exposure point concentration ranges were selected based on typical levels reported in the literature, many of which were summarized in the draft EPA report on indoor air pollution. They were selected to reflect the average levels one might find if a particular source were present. We have tried to exclude pollutant levels representative of worst-case situations from our analysis.

Because each pollutant evaluated can be associated with a particular source and because individuals without exposure to a pollutant source are likely to have a much lower risk, the population at risk depended on the pollutant and the number of individuals exposed to that particular source or demonstrating a certain behavior. Additionally, for the non-cancer health effects, the population at risk was often a subset of those individuals potentially exposed to any given source, usually those who are more susceptible to the health effects of concern. U.S. census information served as a basis for much of the population data. The estimated population for the six New England states is approximately 12 million. The estimated figure representing the civilian labor force is 6 million, and approximately 2 million people in New England are in school grades K through 12.

Risk Characterization

The compounds associated with the greatest individual risk for cancer were environmental tobacco smoke, chlordane, phthalate esters, formaldehyde, and heptachlor. These compounds were also associated with the greatest population risk. Exposures in the home environment contributed most significantly to the overall cancer risk estimate. The total estimated individual cancer risk from exposure to all pollutants evaluated ranged from 0 to 1E-2, and the total population cancer risk estimate for all nine pollutants evaluated ranged from 0 to 1E-3. The corresponding annual incidence is between 0 and 50.

For the evaluation of chronic non-cancer effects, estimated exposures to CO, NO_x, and CH_2O only slightly exceeded the standard and thus each received a low ratio score. The health effects of concern at the respective standards varied in severity from mild to moderately severe. In general, large populations were deemed to be potentially exposed to the pollutant levels evaluated.

Acute non-cancer effects were also evaluated for CO, NO_x, and respirable particulates. The severity and potency scores remained unchanged from the chronic evaluation but a smaller population was considered at risk. The acute effects did not warrant a change in our thinking on the importance of the non-cancer health effects.

One source of uncertainty is the conservative approach taken in assuming that people may actually be exposed to every pollutant evaluated at the levels reported. Anyone we could associate with a certain behavior or source was assumed to be exposed to the average levels reported. Estimates of sensitive populations and populations at risk (demonstrating a certain behavior or having a particular source) proved to be very difficult. Consequently, much of the population information was collected from census statistics. Because we had very little monitoring information from the Regional Office, levels of exposure were based on typical concentrations found in indoor environments from studies done across the nation. While not specific to Region I, we felt the levels probably do not change radically from one location to another. Non-carcinogenic health effects at levels below standards were not considered in this evaluation.

We felt the percentage of problem covered was high because of the relatively large number of pollutants covered. We did not evaluate every indoor air pollutant for the problem area but we tried to select those pollutants that represented different sources and chemical groups, and ones for which we had exposure information.

6. Radiation from Sources Other than Radon

Problem Area Definition

This problem area includes risks from exposure to radiation other than indoor radon. Various sources of ionizing radiation (such as occupational exposure, nuclear power plants, and low level radioactive wastes) were eliminated from this problem definition because of the lack of regional EPA involvement in managing them. The problem area was then expanded to include sources of non-ionizing radiation. Although regional involvement in these sources might also be limited, it was thought that the potential for generally unrecognized risks should be explored through the Risk Reduction Project.

Two general sources of non-ionizing radiation are of concern in non-occupational settings: radio frequency radiation (mainly FM radiation in the vicinity of FM antennas), and extremely low frequency (ELF) radiation--the electromagnetic radiation that is found in the vicinity of 60 Hz power sources. ELF has recently been the subject of both laboratory research and epidemiological studies. Because of the pervasiveness of ELF and the availability of some quantitative estimates of risk from ELF, it was decided to analyze this problem in terms of risks from ELF radiation. It should be recognized, however, that there are other sources of non-ionizing radiation and ionizing radiation that are not considered in this evaluation. Regional data were not evaluated for this problem area. Rather, this paper discusses the implications of current research on the risk posed by ELF radiation.

Summary/Abstract

The most comprehensive review of the effects of powerlines is the New York State Power Lines Project (July, 1987). This $5 million project reviewed existing research and funded additional studies to evaluate a large variety of possible health effects that could be caused by electric and magnetic fields resulting from transmission lines. Studies included an evaluation of teratogenic effects, effects on cell biology, effects on the proliferation of cancer cells in culture, neurobiological effects, behavioral effects, multidisciplinary studies of human exposure, and the epidemiology of cancer incidence.

Several areas of possible concern were identified, but a study by Savitz that demonstrated a possible association between residential magnetic fields and the incidence of childhood leukemia in Denver was of particular concern. In the Savitz case-control study, all cases of childhood cancer (ages 3-14) between 1978 and 1983 were evaluated and compared with a control group. Exposures to magnetic fields were estimated by determining wiring configurations and by direct measurement of fields. Results showed that there was an association between wiring configuration and increased cancer risk. This held for all childhood cancers, especially for leukemias, and, to a lesser degree, for brain tumors. The relative risk was 2 for the highest exposed group.

According to the study, the incidence rate for childhood cancers is about 1 per 10,000 children per year. If the relationship shown by Savitz is true, then the risk for the most exposed individuals would be increased to 2 per 10,000 children per year. The study concludes that this could mean that 10 to 15 percent of childhood cancers are attributable to magnetic fields. An EPA estimate of the number of cases nationally (using the 15 percent risk estimate) is 300 cases per year attributable to ELF.

Although the Savitz study is the most conclusive epidemiological study conducted, there have been others as well. These studies were criticized because they measured only powerline proximity and not the actual fields in the homes. More definitive analytical epidemiological studies are necessary to strengthen this potential association.

Toxicity Assessment

The "pollutant" in this case is the magnetic field that is generated by 60 Hz powerlines. The mechanism of causation is not known, but it is possible that these fields may promote cancer by stimulating growth-related hormones in cancerous cells. The cell-stimulating effects of electromagnetic fields have been studied for years in efforts to increase cell activity to heal bone fractures. In a separate powerlines project study, researchers reported that cancer cells reproduced faster after exposure to electromagnetic fields.

Exposure Assessment

Fields generated by 60 Hz powerlines are ubiquitous, and are generated by any type of powerline, from large transmission lines to smaller distribution lines to household wiring. Magnetic fields are measured in units of the tesla or the gauss. In the Savitz study, the risk of leukemia increased with measured magnetic fields, with the relative risk of getting leukemia at 2.1 for fields greater than or equal to 0.25 microtesla (2.5 milligauss).

An extrapolation from the Savitz study to the rest of the country would be based on the assumption that the exposure patterns found in the Denver area would be the same everywhere else. The population studied by Savitz was not large--only 357 cases.

The population at risk is defined to include children between the ages of 3 and 14.

Risk Characterization

The risk estimates derived from the Savitz study indicate that the relative risk of childhood cancer is twice as great for children in high-exposure homes. The risk increases from 1 in 10,000 to 2 in 10,000.

The uncertainty about these risk estimates is high. However, the estimates do form the basis for a hypothesis that childhood cancer is promoted by exposure to ELF fields.

The overall estimate on the percentage of the problem covered is medium.

Issues not considered include the following:

- Effects of ELF on adult populations
- Effects of RF fields
- Effects of ionizing radiation from sources not considered in the problem definition

7. 8. & 9. Industrial Point Source, POTW, and Nonpoint Source Discharges to Surface Waters

Problem Area Definition

The three problem areas dealing with discharges to surface water were combined for this analysis. Because contamination may be the result of discharges from several different sources, it is difficult to clearly separate these categories and attribute problems to a particular source.

The sources considered in this analysis include point source discharges from direct industrial dischargers; discharges from POTWs that may include wastewater from municipal sewage treatment systems, wastewater from industrial indirect dischargers, and combined sewer overflows; and nonpoint discharges that do not originate from a discrete conveyance or pipe but may be due to runoff, discharge from contaminated ground water, and suspension or dissolution of contaminated sediments.

Summary/Abstract

Using data on fish contamination in areas where fish advisories are in effect, we calculated a cancer incidence rate of 16 to 32 cases annually due to PCB contamination. The individual exposure risk is approximately 7.6E-3 to 1.5E-2. The population exposed is 150,000, an estimate of the number of recreational fishermen in the region. For non-cancer risks, the estimated doses for lead and mercury are 10 times and 25 times the oral RfD, respectively. This relatively high cancer and non-cancer risk is attributed predominantly to discharges from industrial and nonpoint sources. From the information available, POTWs did not contribute significantly to the contamination considered and therefore received a lower ranking.

Toxicity Assessment

The major chemicals of concern in this analysis include lead, mercury, PCBs, and dioxin. These are the substances on which fish consumption advisories have been based in Massachusetts and Connecticut. The RfDs (mg/kg/day) for the non-carcinogens are 6E-4 for lead and 5.1E-5 for chronic effects from mercury. The health endpoints associated with lead and mercury include increased blood pressure, reproductive effects, and central nervous system effects. The cancer potency factors used in the analysis include 7.7 per mg/kg/day for PCBs and 1.56E-5 per mg/kg/day for dioxin.

Exposure Assessment

As discussed previously, there are numerous potential sources of contamination; the specific type, size, and location of sources may not be readily identified. Our information does, however, allow us to draw some conclusions about the relative importance of industrial, POTW, and nonpoint sources.

The primary pathway of exposure considered in this analysis is ingestion of contaminated fish from surface waters. The health risks associated with inhalation and direct contact with surface water are assumed to be minimal. A real risk may be associated with the direct ingestion of surface water. However, this risk is difficult to assess. Because the population consuming untreated surface water is small and water concentrations of contaminants are low, the risk is assumed to be significantly less than that associated with fish ingestion.

Several assumptions were used in characterizing the exposure to contaminants. We assumed that there is no risk from areas in which fishing has been legally restricted. We only considered areas where people might continue to fish even though fish consumption advisories are in effect. Exposure calculations were based on average concentrations of contaminants in fish from data in Massachusetts and Connecticut. In cases where average concentrations across sampling stations or species varied significantly, we used a range of the average measured concentration.

The exposure assessment also uses a typical consumption figure of 46 grams/day that is based on the average consumption of fish among Caucasian marine sport fishermen and women in New England, a statistic from the National Marine Fisheries Service. We assumed that the consumption by non-marine recreational fishermen and women would be roughly the same.

We calculated that approximately 1.5 million people in Region I fish recreationally. This number is based on data from the National Marine Fisheries Service and on a survey. Of this population, however, we assume that only 10 percent of the recreational fishermen and women would be fishing in areas where contamination may be a problem, i.e., areas where fish consumption advisories are in effect or could potentially be issued.

The average concentration values and estimated consumption value allowed us to calculate typical doses (mg/kg/day) of contaminants:

$$\begin{array}{c} \text{Dose} \\ \text{(mg/kg/day)} \end{array} = \begin{array}{c} \text{Conc. in} \\ \text{fish tissue} \\ \text{(mg/kg)} \end{array} \times \begin{array}{c} \text{Average} \\ \text{consumption} \\ \text{(kgs/day)} \end{array} \quad \begin{array}{c} \text{divided} \\ \text{by} \end{array} \quad \begin{array}{c} \text{Average} \\ \text{body weight} \\ \text{(70 kgs)} \end{array}$$

Risk Characterization

Using these exposure assumptions, we calculated cancer and non-cancer risk estimates. The cancer risk estimate associated with PCBs is 7.6E-3 to 1.5E-2 (using a range of the average concentrations of PCBs in fish). The cancer risk from dioxin is minimal (1E-9). The individual cancer risk for PCBs translates into a population risk of 1,140 to 2,250 cancer cases over a lifetime. Using a 70-year average lifespan, the annual cancer incidence associated with PCBs is 16 to 32 cases.

The non-cancer risk from ingestion of contaminated fish is due to consumption of lead and mercury. The typical dose of lead is calculated to be 5.8E-3 mg/kg/day. This is approximately 10 times the RfD for lead. For mercury, the typical dose ranges from 3.3E-4 to 1.3E-3 mg/kg/day. The upper range of this dose is approximately 25 times the RfD for chronic effects. The health endpoints associated with lead and mercury include increased blood pressure, reproductive effects, and central nervous system effects. These are assigned a severity score of 3. The population score corresponding to 150,000 people is 3. Finally, the potency score associated with doses ranging from 10 to 25 times the RfD is 2. The total score for non-cancer therefore is 8.

These risks are assumed to be associated predominantly with contamination from industrial discharges and nonpoint sources. The contribution from POTWs is assumed to be minimal, thus the score for POTWs is lower than that for industrial and nonpoint sources.

Based on this analysis, the uncertainty in the risk estimates is considered to be medium. The percentage of the problem covered, however, is assessed as low because only a small number of pollutants was evaluated.

10. Discharges to Estuaries, Coastal Waters, and Oceans from All Sources

Problem Area Definition

This problem area assesses risks from exposure to contaminants discharged to estuaries, coastal waters, and oceans from all sources. These sources may include runoff, ocean sludge disposal, combined sewer overflows, POTW wastewaters, air deposition, and others.

Summary/Abstract

Using data on contaminant concentrations in shellfish and finfish measured throughout New England, we calculated a total individual cancer risk of 4.9E-4. This cancer risk represents the sum of individual risks from nine contaminants. Risks from individual chemicals range from approximately 2E-4 for heptachlor and PCBs to 5E-8 for DDT and hexachlorobenzene (HCB). Assuming the total New England population of 12.8 million people is exposed, this risk would translate into approximately 6,272 cancer cases over a lifetime (70 years). The annual incidence is therefore 90 cases.

Non-cancer risks are not expected. None of the 18 pollutants assessed exceeded their respective reference doses (RfDs).

Toxicity Assessment

The data we examined included 18 different pollutants: PCBs, silver, cadmium, chromium, copper, iron, mercury, nickel, lead, zinc, DDT, dieldrin, heptachlor, lindane, PAH, chlordane, hexachlorobenzene, and hexachlorocyclohexane. The cancer potency factors for these pollutants are listed in Table 10-1, along with the RfDs, typical concentrations, and calculated doses. The doses calculated are all significantly below the oral RfDs, with the exception of lead. The exposure dose for lead approached one-half the RfD. The various non-cancer health endpoints associated with the RfDs were therefore not addressed.

Exposure Assessment

There are numerous potential sources of contamination; the specific type, size, and location of sources cannot be readily identified. The levels of contaminants found in shellfish and bottom fish generally correlate well with sediment concentrations. Sources of sediment contamination are not easily identified but often result from industrial point sources, CSOs, or wastewater discharges. This analysis did not attempt to attribute contamination to particular sources.

Table 10-1

Estimated Risk Associated with Fish Contamination

	Concentration in Fish (mg/kg)	Typical Dose (mg/kg-day)	C.P.F. $(mg/kg\text{-}day)^{-1}$	Cancer Risk	RfD (mg/kg-day)
PCBs	0.1	2.3E-5	7.7	1.8E-4	1.0E-4
Silver	0.03	7.0E-4			3.0E-3
Cadmium	0.2	4.6E-5			2.7E-4
Chromium	0.4	9.1E-5			5.0E-3
Copper	3.0	6.9E-4			3.7E-2
Iron	35.0	8.0E-3			
Mercury	0.03	7.0E-6			2.0E-3
Nickel	0.3	6.9E-5			
Lead	1.0	2.3E-4			1.4E-3
Zinc	25.0	5.7E-3			2.1E-3
DDT	0.006	1.4E-7	0.34	5.0E-8	5.0E-4
Dieldium	0.02	4.6E-6	16.0	7.3E-5	
Heptachlor	0.2	4.6E-5	4.5	2.1E-4	
Lindane (g-HCH)	0.002	4.6E-7	1.3	5.9E-7	3.0E-4
PAH	0.01	2.3E-6	11.5	2.6E-5	
Chlordane	0.003	6.9E-7	1.3	8.9E-7	5.0E-5
HCB	0.00013	3.0E-8	1.67	5.0E-8	8.0E-4
HCH	0.00015	3.0E-8	6.3	2.2E-7	3.0E-4

The only route of exposure considered in this evaluation was ingestion of contaminated fish and shellfish. Some risk may also be associated with direct contact or swimming in contaminated water; however, this is considered minimal in comparison with risks from ingestion.

Several assumptions were used in characterizing the exposure to contaminants. We used "typical," or median, concentrations of pollutants, based on data from various studies summarized in a report by the Woods Hole Oceanographic Institute and the Environmental Science Program at the University of Massachusetts. This report contains measurements of fish tissue and shellfish concentrations for a number of contaminants, species, and sampling locations throughout New England. We have also used contaminant levels from a separate report on Quincy Bay. In Quincy Bay, the typical fish concentrations listed for cadmium, chromium, copper, mercury, lead, and DDT were all slightly less than the concentrations we chose as "typical" from our other New England data. The mean levels of PCBs and PAHs, however, were higher than the median concentrations listed in the New England data. For all concentrations, therefore, we relied on typical concentrations from the New England data, except in the case of chlordane, hexachlorobenzene, and hexachlorocyclohexane, for which we only had concentration data from Quincy Bay.

It is important to keep in mind that when we determined typical or median concentrations, we excluded data taken from New Bedford Harbor, since all fishing and shellfishing in the harbor has been banned. In this area, concentration observed may not be considered as representative of the exposure concentration. This may or may not be an accurate assumption. It is assumed in this analysis that other measurements of concentrations from the New England waters represent the typical concentrations observed in Region I.

The typical exposure dose of the contaminants was calculated by using a typical consumption rate of 16 grams/day for the New England population. This rate represents an average consumption for all the people in New England, as determined by the National Marine Fisheries Service. The typical exposure doses calculated for an adult (70 kg body weight) are shown in Table 10-1. The population exposed is assumed to be the total New England population, or 12.8 million people.

Risk Characterization

Cancer risk estimates associated with exposure to several pollutants are presented in Table 10-1. Assuming that cancer risks should be additive, the total individual cancer risk is calculated as 4.9E-4. This translates into a total of 6,272 cancer cases over a lifetime (70 years) for the entire New England population, or an annual incidence of 90 cases. Non-cancer risk is not expected to occur.

This estimation of annual cancer cases probably represents an upper bound estimate. Calculation of the median concentrations may involve some hot spots of contamination. The uncertainty associated with these estimates is high. The percentage of the problem covered, however, is medium, because of the number of contaminants considered.

12. Drinking Water

Problem Area Definition

As drinking water arrives at the tap, it may contain a wide variety of contaminants from both natural and man-made sources. This category covers water supplies from both surface water and ground-water sources. It relates to watershed management and drinking-water contamination from the source to the tap. Exposure may be through ingestion, inhalation of volatilized contaminants, and dermal contact. It excludes contamination from waste sites (problem areas # 13, # 14, # 15, # 16) and storage tanks (#18). The impact of lead in drinking water is evaluated in the Lead problem area (#22) and that of radon from drinking water is evaluated in the Radon problem area (#4).

Summary/Abstract

Individual cancer risk derived from exposure to drinking water contaminants evaluated in this analysis is approximately 2E-4. The annual excess cancer cases attributable to drinking-water contaminants are estimated to be lower than 20. Doses rarely exceed the Reference Dose (RfD) or Maximum Contaminant Level (MCL). When they do, it is on the order of one to five times the benchmark. This analysis is based on monitoring data, data on violations of MCLs from the Federal Reporting Data System (FRDS), and information from the Federal Register (Advanced Notice of Proposed Rulemaking for Radionuclides).

The quality of the data used in this analysis is considered inadequate. The percentage of the problem covered is medium and the uncertainty is high.

Toxicity Assessment

The primary chemicals of concern identified for this problem area in Region I are some of those contaminants regulated or considered by EPA for regulation. These include arsenic, copper, microbial contamination, total trihalomethanes (TTHMs), nitrates, and radionuclides (excluding radon).

Major sources of pollutants evaluated are:

- Natural sources for arsenic and radionuclides.
- Corrosion of potable pipings and joints by corrosive water for copper and lead. Although lead is also a major contaminant of concern in Region I, it is evaluated in the Lead problem area (#22).
- Point sources from effluent discharges, sewer overflow, sewage treatment plants, and failure of septic systems and nonpoint sources for microbial contaminants.
- Application of lawn-care or agricultural fertilizers and failure of septic systems for nitrates.
- Disinfectant byproducts from chlorination for TTHMs.

Of the contaminants evaluated, arsenic and radionuclides are human carcinogens (Class A). Chloroform is the predominant compound (>80 percent) among the TTHMs derived from chlorination of drinking water. Chloroform is considered a probable human carcinogen (Class B2). The Cancer Potency Factor (CPF, or slope factor) used for this analysis (see Table 12-1) is assumed to be the same as that derived for chloroform (8.33E-6 per mg/L). The CPF for arsenic is 4.29E-5 per mg/L, and the CPF for radium-228 is 8.8E-6 per pCi/L.

For non-carcinogenic effects, the reference dose (RfD) is used in this evaluation. Where the RfD is not applicable or available for the contaminants, the relevant MCL is used. The RfDs for copper, chloroform, and nitrates are 0.037 mg/kg/day, 1 mg/kg/day, and 0.01 mg/kg/day, respectively.

Other contaminants were evaluated against their MCLs as promulgated by EPA. The MCL for arsenic is 0.05 mg/L, and the MCL for coliform is 1 count per 100 ml sample analyzed. There is no MCL for Giardia. The toxicity endpoints associated with the contaminants are shown in Table 12-1 along with their respective CPF, RfDs and MCLs.

Table 12-1

Toxicological Parameters of Drinking Water Contaminants

Contaminants	Carcinogenicity Classification	Cancer Potency Factor	Reference Dose	MCL	Toxicological End Points
Arsenic	A	4.29E-5 (ug/L)-1	-	0.05 mg/L	Cancer
Copper	-	NA	0.037 mg/kg/day	-	GI irritation
Nitrates	-	NA	1.0 mg/kg/day	10 mg/L	Methemoglobinemia
TTHMs*	B2	8.33E-6 (ug/L)-1	0.01 mg/kg/day	100 ug/L	Cancer
Radium-228	A	8.8E-6 (pCi/L)-1	-	5 pCi/L	Cancer
Coliform	-	NA	-	1 count per 100 ml.	GI Irritation
Giardia	-	NA	<1 cyst		GI Irritation

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* Use chloroform as the surrogate compound. CPF and RfD derived from ingestion of chloroform administered in drinking water is used.

- Not available.

NA: Not applicable.

Exposure Assessment

The data sources for evaluating exposure to drinking-water contaminants varied. Monitoring data are available from state agencies or their laboratories, but due to time constraints and resource limitations this evaluation only used data that were easily accessible. Information from the FRDS for MCL violations during 1987 were examined. The only violating parameter was coliform. Although there were rarely MCL violations for TTHMs, Region I nonetheless is concerned with measured concentrations below MCL of 100 ug/L. For this analysis, we examined TTHM monitoring data compiled by Region I's Water Supply and Ground Management Branch from data submitted by all six New England states. A random sample of 50 systems was selected to compute median values for each state and for New England as a whole. For arsenic, copper, and nitrates, we examined sampling results from a state laboratory in New Hampshire. Radium data were based on the regional information published in the Federal Register, Vol. 51, No. 189, 34836-34862, September 30, 1986. The Giardia information was based on an interview with staff from the EPA Laboratory in Lexington, Massachusetts, and a selection of newspaper and journal articles.

The median concentrations of contaminants, which are considered to be representative of typical conditions, are presented in Table 12-2.

Table 12-2

Estimated Median Concentration of Contaminants Found in Drinking Water

Contaminants	Median Concentration
Arsenic	<5 ug/L
Copper	100 ug/L
Nitrates	0.25 mg/L
TTHMs	49 ug/L
Radium-228	0.10 pCi/L
Coliform	1-5 counts per 100 ml
Giardia	Positive testing

Outbreaks of Giardia were documented in at least four surface-water supply systems in New England. It was estimated that 10 percent of all finished water sampled from surface-water supplies tested positive for Giardia. It should be noted that those tests where Giardia was not isolated from the sample do not guarantee that the water supply was negative for Giardia. It simply means the analyst was unable to isolate Giardia from the sample. Giardia cysts are considered to be a threat to virtually all water utilities using surface waters.

Routes of exposure to contaminants in drinking water include ingestion, inhalation, and dermal absorption. However, the only route of exposure quantitatively evaluated in this analysis is ingestion. Inhalation of TTHMs released from all uses of contaminated water in the household could possibly contribute as much exposure dosage as that due to ingestion. Therefore, the cancer risk estimate was doubled from what was calculated for the ingestion route. Dermal absorption can also contribute additional exposure dose. However, it is believed to be not greater than 25 percent of that contributed by the ingestion route in the worst case. Dermal exposure is not quantitatively evaluated.

The exposed population varied depending upon the contaminants examined (Table 12-3). For TTHMs, the exposed population was estimated to be 7 million (those using surface-water supplies). For arsenic and radium, the population was determined to be 6 million ground-water users. For contaminants where violations were evaluated (i.e., coliform, copper, and nitrates), the exposed population was estimated as only those serviced by water systems with violations. For coliform and copper, there were approximately 40 violating systems across the Region I; the population exposed was determined to fall into the 10,000 to 100,000 range. Similarly, the population was considered to fall in the 1,000 to 10,000 range for children exposed to public water systems violating the nitrates MCL.

A study conducted by the Colorado State University (1986) examined the presence of Giardia in raw and finished surface water and ground water from 300 sites in 28 states. Approximately 10 percent of all finished surface water supplies tested positive for Giardia. Combining this information with the total number of surface water users in Region I (7 million) led to our estimate of 700,000 people exposed to Giardia.

Risk Characterization

In the evaluation of daily exposure doses, a daily ingestion of two liters of water for 70-kg adults for the lifetime of 70 years is generally assumed. For nitrates, one liter of daily water consumption is assumed for 10-kg children.

Chronic daily intakes (CDI) are calculated as follows:

$$\text{CDI} = \frac{[\text{Contaminant concentration}] \times 2\ \text{L}}{70\ \text{kg}}$$

or in the case of children exposed to nitrates:

$$\text{CDI} = \frac{[\text{Contaminant concentration}] \times 1\ \text{L}}{10\ \text{kg}}$$

Table 12-3

Characteristics and Size of Population Exposed to Drinking Water Contaminants

Contaminants	Population Characteristics	Number of People Exposed
Arsenic	Ground-water users	6,000,000
Copper	Users of public water supply violators	10,000-100,000
Nitrates	Children served by public water supply violators	1,000-10,000
Radionuclides	Ground-water users	6,000,000
TTHMs	Surface water users	7,000,000
Coliform	Users of public water supply violators	10,000 -100,000
Giardia	Users of surface water tested positive	700,000

For non-carcinogenic effects, The Hazard Index (HI) is a comparison of CDI to the relevant RfD considered protective of adverse health effects.

$$\text{Hazard Index (HI)} = \frac{\text{CDI}}{\text{Reference Dose*}}$$

* MCL is used for coliform and arsenic.

Assuming additivity, the HI for contaminants with similar modes of action for toxicity or similar toxicity endpoints can be summed.

Incremental lifetime individual cancer risk is a function of CDI and chemical specific CPF.

Individual cancer risk = CDI x CPF

Annual cancer incidents in Region I = Individual cancer risk x number of people exposed to the contaminants in the region/70 years

Table 12-4 presents the estimated HI, the individual lifetime cancer risk, and the annual cancer incidence in New England resulting from exposure to drinking contaminants evaluated in this analysis.

Individual cancer risk was estimated at 1.6E-5 for surface water users, based on the median TTHM concentration (49 ug/L) across the region. Risk from exposure to arsenic was estimated at the detection limit (a limitation of the monitoring data used) for an individual risk of 2.1E-4. Radium risks were estimated using average values for the six Region I states at 8.8E-6.

Table 12-4

Risk Characterization of Drinking-Water Contaminants Examined

Contaminants	HI	Lifetime Individual Cancer Risk	Annual Cancer or Disease Incidence**
Arsenic	<1	2.1E-4	<17
Copper	<1	NA	†
Nitrates	<1	NA	†
TTHMs	<1	1.7E-5	2
Radium-228	†	8.8E-7	<1
Coliform	5	NA	†
Giardi	*	1E-2*	7,000

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*Individual risk for Giardiasis.
** Annual incidence estimated in Region I.
† = Not analyzed
NA = Not applicable .

The population risks were estimated using the exposed populations discussed above. For TTHMs, median concentration values and surface-water-dependent populations specific to each state were used to develop the population risk. The resulting estimate was about 120 cancer cases in 70 years, or approximately two cases per year. Arsenic (at the detection limit) results in an estimate of approximately 17 excess cancer cases per year. Radium exposure results in an estimated less than one cancer case per year. The estimate of total cancer cases is less than 20 per year.

For the non-carcinogenic effects, the estimated CDI are below their respective RfDs. Since these chemicals do not exhibit similar systemic toxic effects, HIs from individual contaminants were not considered additive. The coliform violations in Region I had a median value of five times the MCL. Incidence data from two recent outbreaks of Giardiasis were used to characterize the risks in that area. Both case studies reported an individual disease risk of 1E-2.

The percentage of the problem covered is considered medium and the uncertainty is high.

Although this analysis primarily relied on monitoring data and captured most of the contaminants of concern in this region, the accessibility of the data is limited. Also, the quality of the data is not uniform across the contaminants examined.

While arsenic, copper, and nitrates frequently present problems for private wells, data for these compounds came from public water supply systems. In addition, extrapolation from data presented in one state (New Hampshire) to the entire region presents a great uncertainty.

It should also be noted that lead, radon, and contaminants released from leaking underground storage tanks are explicitly excluded from the analysis for this problem area in order to conform to the definition for this specific problem area. In Region I, leaking underground storage tanks have been identified as one of the major contributors to private well contamination. Lead found in drinking water, primarily as a result of corrosion from delivery piping systems, also causes concern.

Radionuclides other than radium are not analyzed. Uraninum data are lacking for this analysis. The analysis of radionuclide data in drinking water, excluding radon and uranium, is only a very small portion of the radionuclide problem.

13. RCRA Waste Sites

Problem Area Definition

This category includes the risks posed by hazardous waste sites regulated under the Resource Conservation and Recovery Act (RCRA). More specifically, it includes operating and inactive RCRA landfills and surface impoundments, hazardous waste storage tanks, hazardous waste burned in boilers and furnaces, hazardous waste incinerators, and associated solid waste management units. Seepage and routine releases from these sources contaminate soil, surface water, and ground water, and pollute the air. There is potential double-counting with risks estimated in Hazardous/Toxic Air Pollutants (problem area #3), and Discharges to Surface Waters (problem areas #7, #8, #9).

Summary/Abstract

The RCRA problem area poses relatively low individual cancer risk (1E-6) and represents approximately 1 to 10 cancer cases per year. The cancer range derives from a rough estimate of the possible impact of solid waste management units (SWMUs). The percentage of the problem covered and the uncertainty are high. The uncertainty is associated with the use of modeling results to estimate ambient concentrations. Non-cancer risks were estimated to be low since no releases from sites were estimated to result in doses greater than the RfD.

Toxicity Assessment

Based on estimates of typical constituents of the hazardous wastes generated in Region I, more than 100 compounds could be expected to be found at RCRA sites across six states. We evaluated cancer and non-cancer endpoints for 18 of these compounds, including arsenic, benzene, cadmium, carbon tetrachloride, chloroform, chromium, dichloromethane, mercury, PCBs, toluene, vinyl chloride, and xylene. The endpoints of concern for these compounds vary from cancer to a variety of systemic effects, including reproductive effects and neuro-behavioral effects.

Exposure Assessment

There are more than 5,200 hazardous waste generators in Region I and more than 228 treatment, storage, and disposal facilities (TSDs). Most of the TSDs in Region I are storage facilities or treatment and storage facilities, with only one operating land disposal facility. Most of the waste generated in the region is shipped to other parts of the United States or to Canada for final disposal. Very little waste is imported into Region I for handling. As a result, risks to the population in New England are primarily from tank and drum storage, treatment, and waste previously disposed of in landfills.

Risk was assessed using the Regional Hazardous Waste Planning Model previously developed by Temple, Barker & Sloane, Inc. (TBS) for EPA. The model was designed to assess the relative costs and risks associated with various hazardous waste management strategies (e.g., increased use of incineration). In estimating risk, the model uses generator-specific data on quantities of waste handled, management practices, location, average release algorithms, and data on exposure parameters. The waste data come from validated biennial reports filed with each state by the generators. The waste constituent data and release algorithms were developed by the Office of Solid Waste. The exposure parameters (hydrogeological setting, temperatures, population locations, drinking-water source type) were developed by TBS for each geographical area in the region where waste handling takes place. These characterizations are rough in some cases, with each location being classified into one of several possible characterizations for each exposure parameter. For example, there are only three possible stream types to describe any given geographical area.

Release and exposure pathways were evaluated for each waste-handling method. The evaluations considered multiple exposure points, air concentrations at four separate distances (from 1 to 19 kilometers) from the source, ground-water concentrations estimated at 60 meters from the source, and surface-water concentrations estimated at 10,000 meters from the source (surface waters are assumed to be treated before ingestion). The total population at risk varies for each exposure pathway. In all cases, however, it is more than one million people (category 4 in our scoring system).

Risk Characterization

Average individual cancer risk is on the order of 1E-6 across the entire region. Total population cancer risk is low, approximately one case per year. The majority of this risk is attributed to hazardous waste incineration. If the blending and burning of used oil in boilers and furnaces had been evaluated for this problem area, this risk level would have been higher. The Hazardous/Toxic Air Pollutants problem area evaluated the burning of used oil. No releases from sites were estimated to result in doses greater than the RfD.

The consideration of SWMUs could raise this risk estimate. Based on an examination of RCRA Facility Assessments for nine facilities in New England, more than 200 SWMUs or an average of more than 20 SWMUs per site were found. These SWMUs were generally of two types: tanks and drums or unlined landfills and lagoons. Further investigation found that the materials in these SWMUs were generally consistent with current activities and wastes at the site.

Assuming that the waste types and annual volumes are roughly equivalent to the present activities at the sites, we developed a very rough estimate of the impact of the SWMUs. The drums and tanks currently regulated pose very minimal risk as estimated by our model; we estimate that the SWMU drums and tanks probably pose a higher but still minimal risk. The SWMUs that are unlined landfills and lagoons, however, may pose risks one to two orders of magnitude higher than the risks posed by currently regulated RCRA landfills. This higher risk is due primarily to the large number of landfill SWMUs. These SWMUs could therefore add another 1 to 10 cancer cases per year to the risk estimated for this problem area.

The Public Health Risk Work Group estimated the percentage of the problem covered as high (all RCRA sites, many pollutants, total waste volume, rough SWMU estimate) and the degree of uncertainty as high. The high uncertainty derives from the use of modeled concentrations rather than monitoring data.

14. Superfund Waste Sites

Problem Area Definition

This problem area includes waste disposal sites that are regulated by Superfund. Generally they are inactive and abandoned. It includes sites on the National Priorities List (NPL), those deleted from the NPL, those that are candidates for the NPL, and any additional sites that states may be addressing. These sites are typically characterized by the presence of hazardous waste and the absence of any current manufacturing, treatment, storage or disposal activity. These sites may contaminate ground and surface water, sediments, and soil, and may pollute the air.

Summary/Abstract

Superfund sites present an increased individual cancer risk in the 1E-5 range and a population risk of less than one case of cancer per year in Region I. Estimated doses are rarely expected to exceed the RfD, and when they do they are generally on the order of 5 to 10 times the RfD. This assessment is based on a detailed analysis of the Endangerment Assessments (EAs) prepared for 13 Superfund sites. The results from the 13 sites were extrapolated to cover the approximately 1,570 sites in Region I that fit this problem category. Both the portion of the problem covered and the uncertainty (due to the extrapolation of results) are considered high.

Toxicity Assessment

The chemicals of concern vary from site to site depending on the particular activities or wastes disposed at the site and the environmental and exposure factors at the site. Approximately 25 Superfund risk assessments were reviewed, but only 13 were considered comparable and therefore evaluated in depth. Documents were not considered comparable primarily because maximum rather than average concentrations had been used in the EA analysis. Of the 13 NPL sites examined in detail, 36 different chemicals were considered in the Endangerment Assessments. The most frequently considered chemicals were vinyl chloride, tetrachloroethylene, toluene, methylene chloride, benzene, PCBs, trichloroethylene, and dichloroethylene. The driving pollutants in the health risk analysis were PCBs, tetrachloroethylene, benzene, vinyl chloride, chloroform, bis-2-ethylhexyl phthalate, benzo-a-pyrene, trichloroethylene, and arsenic. These pollutants occurred in concentrations and exposure pathways that dominated other pollutant/concentration/ exposure combinations at the 13 sites examined in detail. The Cancer Potency Factors (CPFs) for these pollutants range from 0.052 $(mg/kg\text{-}day)^{-1}$ for tetrachloroethylene to 4.34 $(mg/kg\text{-}day)^{-1}$ for PCBs. The only pollutants for which an RfD was exceeded were dichloroethylene and tetrachloroethylene. The health effects associated with these RfDs are hepatic lesions for dichloroethylene and liver and kidney changes for tetrachloroethylene.

Exposure Assessment

There are currently 59 sites on the NPL list in Region I (as of early June 1988) and a total of approximately 1,700 sites that are either on the NPL list, are candidates for the NPL list, or are being addressed by the six New England states. Approximately 130 sites require no further action, thus leaving approximately 1,570 sites (59 of which are current NPL sites) that fit this category. The sites represent a variety of activities (manufacturing, municipal waste disposal, hazardous waste storage/recycling, etc.) and are located throughout the region in both rural and urban areas.

For this analysis, we examined recently completed EAs for 13 Region I NPL sites. The results from these sites were used to characterize the risks at all NPL sites and ultimately at all 1,570 sites in this problem area.

The risks estimated represent the risk posed to an "average exposed individual"; reasonable exposure pathways and mean concentrations, rather than maximum concentrations reported in monitoring data, were used. We used the information in the EAs (e.g., doses, concentrations) to estimate risks associated with reasonable exposure pathways, i.e., pathways that could be expected to result in human exposure now or in the future and that had not already been controlled. As a result, worst-case scenarios, such as future construction of a residential well in the middle of a disposal site, were not included, but offsite exposures and risks from trespassing were.

It is important to note that these pathways and risks are not necessarily those selected in the EAs as representative of "current" or "future" risks, because the EAs often evaluate the risk from developing the site for residential use. Also, we selected the highest risk pathway of all the "reasonable" pathways evaluated at a site to represent the risk. That is, we did not add risks from more than one pathway to arrive at an aggregated individual or population risk.

The exposure pathways examined at each site varied. Seven sites evaluated ground-water ingestion as the pathway of concern; two examined dermal exposures to trespassing children or teens; three examined inhalation exposures; and one examined the risks from fish ingestion.

The populations at risk at each site were estimated from information in the EAs. The population is that associated with the particular pathway selected to represent the site. It was often very roughly estimated. The exposed populations at the 13 sites evaluated varied from 25 for ground-water ingestion at one site to 1,500 for ground-water ingestion at another site.

Risk Characterization

In order to determine if our analysis of the 13 sites could be considered in any way to be representative of the other 46 NPL sites, we examined two pieces of information. First we computed the average Hazard Ranking System (HRS) score for all 59 NPL sites (45) and compared it to the average HRS score for our sample (40). Although the HRS ranking is a rough ranking, we felt we could conclude that our sample did not appear to include sites that were significantly more or less risky than the total universe of NPL sites in Region I. We also determined that the site type profile of our sample was not significantly different from the 59 NPL sites as a whole (see Table 14-1). As a result, we felt confident that our results would not be radically different from results obtained for the population as a whole.

Table 14-1

Distribution of Superfund Site Type

Sites	Sample	Region I NPL
Private Landfills	5	15
Municipal Landfills	3	8
Manufacturing Operations	2	12
Hazardous Waste Storage/Recycling	2	11
Other	1	13
Total	13	59

After much discussion in the work group, it was decided that the other non-NPL sites could be regarded as being of similar risk. There was no reason to assume that those sites were any more or less risky then the present NPL sites. As a result, it was felt that the sites examined could be used to represent all the sites categorized in this problem area.

The increased individual cancer risk across the 13 sites evaluated ranged from 1.3E-6 to 1.3E-2. The median value for the sites was approximately 1.3E-5. RfDs were exceeded at two of the 13 sites with the Dose/RfD ratio varying between two and nine for tetrachloroethylene and dichloroethylene.

Population risk was estimated by using the median number of people exposed at the 13 sites examined (250 people) and multiplying that figure by the number of sites (1,570) to arrive at the population exposed (approximately 400,000). This number multiplied by the median individual risk (1E-5) resulted in an estimated cancer risk for the problem area of four cancer cases over a 70-year period, or less than one case per year.

Using the non-cancer scoring system discussed elsewhere, the following scores were assigned to this problem area: 1 for individual exposure ratio, 1 for population exposed to non-carcinogens, and 1 for severity score.

The work group rated the uncertainty associated with these estimates as high, based largely on the extrapolation from the small sample size to the large population. The portion of the problem covered was rated as high by the group, based on the fact that numerous chemicals were considered in the evaluation.

15. Municipal Waste Sites

Problem Area Definition

This category consists of open and closed municipal landfills, municipal sludge and waste incinerators, municipal surface impoundments, land application units, and land treatment units. These sources can contaminate ground and surface water, the soil, and air. There is potential double-counting of the risks from this category with those from Hazardous/Toxic Air Pollutants (problem area #3), Discharges to Surface Waters (problem areas # 7, # 8, # 9), and Other Ground-Water Contamination (problem area #19).

Summary/Abstract

The average individual risk associated with exposure to drinking water 600 meters from a municipal landfill is estimated to be on the order of 1E-5. Population risk was not estimated. The percentage of the problem covered is medium because approximately half of the municipal facilities were evaluated. The air exposure risks were not estimated. The uncertainty is high due to the use of modeled concentrations and few data points.

Toxicity Assessment

The contaminants from municipal landfills include a set of approximately 200. The group evaluated for the risk evaluation includes vinyl chloride, arsenic, tetrachloroethane, dichlormethane, carbon tetrachloride, antimony, and phenol. These contaminants were selected due to their concentrations in municipal landfill leachate, toxicity to humans, regulatory limits under SDWA, and mobility and persistence in the subsurface. Five of the contaminants are carcinogens. The non-cancer effects are neurotoxicity, cardiovascular changes, and kidney and liver effects.

Exposure Assessment

The problem definition in Region I includes:

operating landfills	739
closed landfills	1,443
sludge and waste incinerators	20
municipal surface impoundments	2,000

This analysis covers only the operating landfills. We have information on 58 of the 739 operating landfills. Of the landfills in Region I, 75 percent are categorized as small, receiving less than 30 tons of waste per day; 23 percent are medium, with 30 to 50 tons of waste per day; and 2 percent receive more than 500 tons per day. The tons of waste received per day, along with the mean net precipitation, enabled us to calculate the leachate volumes for each category of landfill. The leachate volume was then multiplied by the median leachate concentration of each of the stressors (derived from the Subtitle D Regulatory Impact Analysis).

To obtain the concentration of the contaminants in the ground water, EPA's Liner Location Model was used. The model has several ground-water scenarios; flow field B, with a ground-water velocity of 10 meters per year in the horizontal and 1 meter per year in the vertical, was used most often. The flow field, mobility of the contaminants, and the concentration of the leachate were combined to give the daily emissions of the contaminants at a variety of distances. The 600-meter exposure distance was chosen because our database showed the average distance to a private well to be 409 meters and the average distance to a public well to be 680 meters.

The database of 58 active municipal landfills showed that 43 percent of the landfills had wells within 1 mile. There were 246 wells in total and, assuming that three people drink water from each well, 738 people were potentially exposed. Five percent of the landfills had public wells within one mile, with five wells total. We did not attempt to estimate the total number of people potentially exposed from this water source because we could not determine an average number of people per water supply.

Extrapolating from the 58 landfills to the 2,182 active and inactive landfills, we find that 21,471 wells may be within 1 mile of the municipal landfills. Therefore, potentially 64,413 people may be exposed.

Risk Characterization

The health risk was characterized by converting the drinking water concentration 600 meters from each landfill into a dose. The dose was then multiplied by a CAG score for the carcinogens or compared to the RfD for non-carcinogens. The individual risk associated with each of the 58 landfills was computed by adding the risk associated with the five carcinogens and by examining the highest resulting non-carcinogen dose to RfD ratio. No landfills had dose-to-RfD ratios greater than one. The cancer risk associated with 60 percent of the landfills was 1E-10, 20 percent had risk in the 1E-4 to 1E-5 range, and 20 percent had risk in the 1E-3 to 1E-4 range. The average cancer risk is 1E-5.

The percentage of the problem covered is medium because approximately half of the municipal facilities were evaluated. The air exposure risks from municipal incinerators and gaseous emissions from landfills (H_2S, methane, and vinyl chloride) were not estimated. The uncertainty is high due to the use of modeled concentrations and few data points.

16. Industrial Waste Sites

Problem Area Definition

This category covers industrial waste, including sludges handled in nonhazardous industrial landfills, industrial surface impoundments, land application units, and land treatment units subject to Subtitle D, along with numerous incinerators. Routine and nonroutine releases, solid migration, and runoff may contribute particulates, toxics, biological oxygen demand (BOD), PCBs, and nutrients to air, surface water, ground water, and soil. Risks from the category could be double-counted with Other Ground-Water Contamination (problem area #19), Discharges to Surface Waters (problem areas #7, #8, and #9), and Hazardous/Toxic Air Pollutants (problem area #3).

Summary/Abstract

The Public Health Risk Work Group analyzed this problem area in relation to the municipal waste sites due to lack of data on industrial waste sites. The work group reasoned that the releases emanating from industrial waste sites may contain similar contaminants but at higher concentrations than releases from municipal sites. Therefore, we agreed that the risks associated with industrial sites are also in the range of approximately 1E-5. Industrial sites were ranked in the same group as RCRA, Superfund, and Municipal Waste Sites for cancer and non-cancer risks. Refer to problem area #15, Municipal Waste Sites, for additional information.

17. Accidental Releases

Problem Area Definition

The uncontrolled accidental release of hazardous substances with a potential for individual or group overexposure by any route with potential serious adverse effects is identified as the problem area. Such releases may be from stationary sources or mobile sources.

Summary/Abstract

Acute, serious, localized, non-cancer effects are considered to be of primary importance in the defined problem area. The frequency of such effects and the numbers of individuals likely to be affected in such events are both believed to be relatively small. In 1985, approximately 800 reported incidents occurred throughout the six states in Region I.

The hazards of the major chemicals of concern are high, especially when they are in the gaseous or vapor phases. Chlorine (gas) features in many reported releases from stationary sources such as paper-making facilities; ammonia gas and hydrogen sulphide gas are other common chemicals of concern. All three substances are extremely toxic in any form.

The health effects associated mostly with acute releases of both inorganic and organic substances are both systemic and local. With few exceptions (notably, asbestos and chlorinated organic compounds), cancer is not associated with an acute release.

The most common pathway of exposure involving acute hazardous releases of toxic gases or vapors is inhalation; here, dermal and ingestion routes are generally quite secondary. However, exceptions exist (notably, high concentrations of acetonitrile, acrylamide, phenolic compounds, aniline and some other primary amines with significant vapor pressures at ambient temperatures).

Exposures with the potential for acute adverse health effects, in the case of accidental releases, are believed to be generally restricted. Individuals in the immediate vicinity of the events or who are present within relatively small areas downwind or to the sides of the release are at the highest risk; i.e., the populations at greatest immediate risk, even in high-volume bursts, are best characterized as "immediate area/near neighbor populations."

The risks to those who are acutely exposed to nearby releases may be best characterized qualitatively; no satisfactory statistical basis is evident for quantitatively characterizing such non-cancer risks.

Risks to populations at large, with respect to non-cancer effects, are believed to be generally small. Attenuation of concentrations of toxic releases is oftentimes claimed to be rapid, in terms of both time and distance, by officials reporting on affected sources. This claim, however, is by no means axiomatic. Topography and meteorological conditions can profoundly affect the extent of potential serious effects associated with some releases of highly toxic substances. (Bhopal is a case in point. In this disaster, meteorological conditions coupled with high population densities in areas surrounding the chemical plant played a large part in the catastrophe.) In such situations, however, it can be very difficult to quantify risk with any generally acceptable level of certainty.

Toxicity Assessment

The common major chemicals of concern in this problem area are basic commercial chemicals: strong mineral acids (sulphuric, hydrochloric, and nitric acids), anhydrous ammonia, chlorine, strong mineral bases (sodium hydroxide and aqueous ammonia), and commercial solvents with relatively low boiling points and high flammability (methyl alcohol, toluene). Most of these common chemicals have appreciable toxicity and/or are potentially explosive. In terms of personal risk, inhalational hazards appear to outweigh individual risk of fatality by fire, based on the generally limited database available for consideration here.

Exposure Assessment

Exposures to excessive levels of airborne concentration of major chemicals of concern is believed to be the more likely cause of acute effect(s) in the event of an accidental release. The major chemicals of concern have assigned ceiling limits but do not have "skin" notations in the "Threshold Limit Values" established by the American Conference of Governmental Industrial Scientists.

Risk Characterization

Risks associated with accidental releases are believed to be essentially non-cancer type end-points. Cancer risk with most of the substances that might be released (even if the substance has a high cancer potency score) is believed to be very low with a single episode. In this case, there appears to be no reliable method to quantify cancer risk.

The uncertainties in the characterizations and qualitative assessments provided here and summarily reported on are believed to be small.

Acute episodes of uncontrolled releases of hazardous chemicals are extensively reported upon and receive media attention, even when no personal injuries are associated with the release. Frequency data (e.g., the number of chlorine releases per year) are known with reasonable certainty. However, the numbers of the individuals within known distances of the release sources and who are actually adversely affected are not known at this time with any acceptable degree of certainty. For this reason, risks associated with acute releases are probably best described qualitatively.

18. Releases from Storage Tanks

Problem Area Definition

This category includes releases of petroleum products or other chemicals from tanks that are above, on, or under the ground, tanks owned by farmers, and the fuel-oil tanks of homeowners. Stored products include motor fuels, heating oils, solvents, pesticides, and lubricants that can contaminate ground water with such toxics as benzene, toluene, and xylene. The primary environmental hazard is contamination of ground water and soil.

Summary/Abstract

The releases from storage tanks are likely to pose low individual cancer risk and possible narcotic effects due to inhalation of gasoline and fuel oil through seepage into homes and during showering. The percentage of the problem covered is low due to the evaluation of motor fuel tanks only. The uncertainty is high because of lack of information regarding concentrations, toxicity, numbers of leaking tanks, and people exposed.

Toxicity Assessment

The risk evaluation focused on petroleum releases. There are literally hundreds of constituents in fuel oil and gasoline. The analysis focused on hydrocarbons, benzene, toluene, xylene, and ethylbenzene. We did not directly evaluate cancer risk or non-cancer endpoints from these compounds due to the lack of data. The major health effects follow inhalation. The effects include narcotic effects, polyneuropathy, general anesthetic effects, and depressant effects on the central nervous system. Some of the organic compounds, such as benzene, are carcinogens.

Exposure Assessment

The number of tanks in Region I is estimated at 758,500, with 143,500 being regulated motor fuel and chemical tanks. The remainder of the tanks are residential and non-residential heating oil tanks and farm heating/motor fuel tanks. The amount of motor fuel and heating oil stored in tanks in Region I is about equal, although the number of heating oil tanks is much greater.

The Underground Storage Tank (UST) program estimates that 10 percent of all tanks at gasoline stations are leaking. Applying that estimate to the total number of tanks in New England, there would be 75,850 leaking tanks. The number of leaking motor fuel tanks would be 14,350. However, we used the estimate of 10,202 because this figure is derived from the tanks reported in the state UST programs. Case studies from Maine showed that hydrocarbon contamination in drinking-water wells ranged from approximately 10 ppb to 40,000 ppb. Benzene levels were 0 to 1,400 ppb, toluene 0 to 7,500 ppb, ethylbenzene 0 to 1,800 ppb, and xylene 0 to 8,800 ppb.

Other potential pathways include inhalation through seepage into homes and showering.

Using the exposure levels from the Maine case studies may overestimate the presence of contamination and concentration levels in the drinking water. In Maine, the gasoline may travel farther and faster due to bedrock fissures acting as conduits. Other New England states have less bedrock geology.

Again using data from Maine, we learned that there are 155 leaking tanks resulting in:

Private wells - polluted	219
Private wells - threatened	174
Public wells - polluted	2
Public wells - threatened	5

Extrapolating from the Maine data, we found that there would be 14,414 total contaminated wells in New England, based on the estimated 10,202 leaking tanks. There would be 25,867 threatened and contaminated private wells. Multiplying the number of wells by three people per well, we find 43,242 people potentially exposed to contaminated well water and 77,601 people potentially using water from threatened and contaminated wells. We did not calculate the number of people potentially exposed from public wells because we assumed that the population estimate would still fall into the 3 category in our scoring system.

Our analysis did not focus on home heating oil tanks or farm tanks. Had we analyzed the health risks from these tanks, the number of potentially exposed people would likely have at least doubled.

Risk Characterization

Due to the lack of contaminant-specific data, we did not estimate risk for individuals and the population. The work group estimated that the cancer risk would fall into category 2, given the presence of known carcinogens but at unknown levels. Additionally, we are not aware of any known concentration or range of concentrations where an "average" individual would cease drinking water contaminated with gasoline due to taste and odor considerations. For non-cancer rankings, the group estimated that the severity ranking would be 3 due to the endpoints mentioned above. The population score was 3 and the potency score was not estimated. Overall, the group felt that the non-cancer risks were in Category 3, and are therefore greater than the risk associated with the various waste programs.

The percentage of the problem covered is low due to the evaluation of motor fuel tanks only. The uncertainty is high because of lack of information regarding concentrations, toxicity, numbers of leaking tanks, and population exposed.

19. Other Ground-Water Contamination

Problem Area Definition

An extensive variety of point and nonpoint sources of pollution not addressed in other problem areas also contaminate ground water. For the purposes of this project, only septic systems, road de-icing salts, Class V underground injection wells, and the leaching of agricultural pesticides and fertilizers were assessed in this problem area due to limited databases, resources, and time. Impacts from underground storage tanks, hazardous waste sites, and landfills are covered in other problem areas for this project.

The list of possible contaminants is even more extensive than that of possible sources. Possible contaminants include microbes, nitrates, sodium, chloride, pesticides, and, due to the inclusion of class V underground injection wells, potentially any waste fluid produced by various industries, utilities, and commercial ventures, including toxic organic and inorganic chemicals, heavy metals, and oil and petroleum products.

Summary/Abstract

The greatest potential risk from this problem area is non-carcinogenic, that assessment being based primarily on bacterial and viral contamination from septic tanks and cesspools. The smaller, carcinogenic risk of this problem area is also thought to be driven by septic tanks and cesspools. This was the only source in this problem area for which a quantitative assessment was attempted.

Septic tanks drive the risk in this area primarily because of their widespread, densely located occurrence in much of the region and a high estimated population at risk (60 percent of the 2 million private well users in New England). The other sources considered also pose some combination of carcinogenic and non-carcinogenic risks to a smaller portion of the 2 million private well users. The assessment is highly qualitative because of a lack of data. The data gap is primarily a function of the population assessed, because we considered only private wells and the water-quality data for these wells are lacking in both space and time. Often, data are reported as well-contamination incidents rather than as actual concentration data. Most private wells in the region are probably at risk from at least one of the sources covered here. The uncertainty in this assessment is high, and the extent of the problem covered is low.

Toxicity Assessment

Major ground-water pollutants from septic tasks and cesspools include microbes (e.g., pathogenic bacteria and viruses), nitrates, chloride, methylene chloride (a.k.a. dichloromethane) and 1,1,1-trichloroethane. Major pollutants from road de-icing salts include sodium and chloride. No "priority" pollutants were assigned to the Class V underground injection wells source because of the variety of well types and lack of data. The pollutants from the wells include, potentially, any waste fluid produced by various industries, utilities, and commercial ventures, as well as septic system pollutants and agricultural chemicals.

Major pollutants found in ground water from agricultural pesticides and fertilizers include nitrates, aldicarb, EDB, alachlor, atrazine, oxyamyl, dinoseb, carbofuran, 1,2- and 1,3-dichloropropane.

Table 19-1 identifies the kinds of microorganisms found in domestic wastewater and the associated disease endpoints that drive the non-carcinogenic risk from contamination by septic tanks and cesspools.

Coliform bacteria are used as indicators of contamination by human and/or animal waste because they are routinely shed in the feces. The primary drinking-water standard for coliform bacteria is less than one per 100 ml of water, the absolute level allowable varying with sampling frequency. The assumption is that if coliform are present, then any other microorganism could be, too, and it may take only one microorganism to produce the resultant disease in a human.

Infants are primarily the population at risk from high nitrate levels in drinking water because they do not have the acids in their digestive system to prevent the growth of bacteria that convert nitrates to nitrites. Nitrites reduce the oxygen-carrying capacity of the blood and can cause death by methemoglobinemia, a type of "suffocation."

The carcinogenic potential of contamination by septic tanks and cesspools is due to toxic chemicals that can be released into such systems by household products and septic tank cleaners. Table 19-2 lists the predicted priority pollutants that can occur in household wastewater.

The primary pollutant chosen to estimate some of the carcinogenic potential of ground water contaminated by septage was methylene chloride (a.k.a. dichloromethane). This was selected because it was the only carcinogenic organic chemical noted in the literature that was cited as being found frequently. It was noted as a septic leachate pollutant by Rhode Island in its 1988 Water Quality Assessment report and was the same chemical evaluated for this source of ground-water contamination in the national comparative risk assessment project. Methylene chloride has a carcinogenic potency factor of .0075 $(mg/kg/day)^{-1}$ for oral ingestion and is currently classified as a probable human carcinogen.

Table 19-1

Pathogenic Microorganisms in Domestic Wastewater

(USEPA, June 1987)

Microorganism	**Disease(s) Caused**
Bacteria	
Salmonella species	typhoid, paratyphoid, gastroenteritis
Shigella	bacillary dysentery
Yersinia	gastroenteritis
Mycobacterium	tuberculosis
Leptospira	leptospirosis
Campylobacter jejuni	gastroenteritis
Pathogenic coliforms	gastroenteritis, urinary tract infections
Yersinia enterocolitica	gastroenteritis
Pseudomonas	respiratory and burn infections, diarrhea
Klebsiella	pneumonia, bronchitis
Serratia	respiratory and urinary tract infections, summer diarrhea
Viruses	
polioviruses	poliomyelitis
hepatitis A	infectious hepatitis
echoviruses	respiratory disease, aseptic meningitis, diarrhea, fever
coxsackieviruses	respiratory disease, fever, aseptic meningitis, myocarditis
Norwalk and Norwalk-like viruses	gastroenteritis
rotaviruses	gastroenteritis
adenoviruses	respiratory disease, eye infections
Parasites	
Entamoeba histolytica	amoebic dysentery
Giardia lambia	giardiasis ("backpacker's diarrhea")
Balantidium coli	dysentery, gastroenteritis
Ascaris ova	pneumonitis, intestinal and nervous system disorders
Trichuris	chronic gastroenteritis
Enterobius vermicularis	enterobiasis
Cestode ova	chronic gastroenteritis
Coccidia	diarrhea, toxoplasmosis

Table 19-2

Predicted Priority Pollutants in Household Wastewater
(ISDS Task Force, 1986)

Organics	Inorganics
benzene	arsenic
phenol	cadmium
2,4,6-trichlorophenol	chromium
2-chlorophenol	copper
1,2-dichlorobenzene	lead
1,4-dichlorobenzene	mercury
1,1,1-trichloroethane	zinc
naphthalene	antimony
toluene	silver
diethylphthalate	
dimethylphthalate	
trichloroethylene	
aldrin	
dieldrin	

With road de-icing salts, primary concern is for sodium as a pollutant. Excessive sodium is unhealthy in any diet, but some segments of the population require low sodium diets for health reasons. High sodium levels stress cardiovascular, liver, and kidney conditions and can affect blood pressure. High sodium levels also may be related to hypertension.

The agricultural pesticides listed as primary pollutants for this evaluation are the ones most commonly found in ground water. They have varying toxicity and carcinogenic potentials. Although many have been well studied regarding health effects because of concerns about food residues, their health effects due to ground-water ingestion are less understood owing to long-term, low-level exposure scenarios and a complex array of physical and chemical factors determining their fate and transport to and in ground water. The only concentration data obtained for this assessment were for aldicarb contamination. Aldicarb (trade name TEMIK) can be adsorbed through all routes of exposure, with ingestion being the most significant. It is acutely toxic, interfering with the transmission of nerve impulses. It has not been shown to be carcinogenic, mutagenic, or to cause birth defects. A study in Wisconsin referenced by the Commonwealth of Massachusetts (1986) showed that aldicarb had an effect on the immune system at concentrations less than 10 ppb, which is the Massachusetts interim guideline standard.

Fertilizers are a source of nitrates, the health effects of which are discussed on page 65.

Because of the wide variety of pollutants possible from *Class V underground injection wells*, no specific health concerns were noted for pollution from this source. For the septic and agricultural wells, other discussions in this section can be referenced.

Exposure Assessment

Sources

Obtaining data on the types, numbers, sizes, and locations of the major sources in this problem area requires documented inventory by the states and their local communities. Whereas a town might have one landfill, it could have hundreds of septic systems, many miles of roads that are salted in the winter, numerous unrecognized Class V underground injection wells discharging hazardous and nonhazardous wastes, and some areal extent of agricultural fields where pesticides and fertilizers are applied. The most readily available statistics about the separate sources appear below.

Septic tanks and cesspools include individual, on-site subsurface disposal systems serving fewer than 20 people. Of all ground-water pollution sources (i.e., not just those in this problem area), septic tanks and cesspools rank the highest in volume discharged directly to soils and are the most frequently reported sources of ground-water contamination (Canter and Knox, 1985). The estimated number of septic systems and a calculation of statewide density of systems per square mile, from 1980 Census of Housing data, appear in Table 19-3.

The total number of septic tanks and cesspools for the region in 1980 was about 1,480,500. Recent estimates by some of the New England states show an increase since 1980: 26 percent for Maine (now about 250,000 systems), more than 9 percent for Massachusetts (now more than 600,000 systems) and 28 percent for Rhode Island (now about 143,900 systems). It is probably reasonable to assume that there are about 1.75 million septic tanks and cesspools in New England. Table 19-4 indicates some county-level statistics, also based on 1980 Census data, which were calculated assuming a uniform distribution of systems within the county.

Septic tanks and cesspools are located throughout the region in unsewered areas. An assumption that they occur only in very rural settings would be erroneous, as can be seen by examination of the counties listed in Table 19-4, which are among the most populous in the region. If the design life of these systems is 10 to 15 years, we can expect that we are seeing the beginning signs of a problem that could only get worse as development in New England increases, because many of the septic tanks and cesspools in New England were installed in the 1960s. It is estimated that only 40 percent of all septic systems function properly, and areas with more than 40 systems per square mile can be considered to have potential contamination problems (Canter and Knox, 1985). Reference to Tables 19-3 and 19-4 shows that the more populous southern New England area have double and even triple this density in some counties.

Table 19-3

1980 Septic Tanks and Cesspools

State	Total Number of Septic Systems (1980)	Systems Per Square Mile
Connecticut	357,446	73.4
Maine	198,629	6.4
Massachusetts	550,629	70.4
New Hampshire	161,386	17.9
Rhode Island	112,663	106.8
Vermont	99,752	10.8

Source: U.S. EPA, January, 1987.

Table 19-4

1980 Septic Tanks and Cesspools/Density

County, State	Systems Per Square Mile
Fairfield, CT	79-158
Hartford, CT	68-135
New Haven, CT	82-164
Bristol, MA	90-180
Middlesex, MA	61-122
Norfolk, MA	127-254
Plymouth, MA	76-153
Worcester, MA	33- 66

Source: Canter and Knox, 1985.

Massachusetts, with more than 600,000 systems and noted surface water problems, appears to be at the greatest risk. On Cape Cod alone it has been estimated that approximately 63.8 million gallons per year (mgy) of septage is generated and only 31 percent treated effectively, leaving about 44 mgy to discharge to a highly vulnerable environment (CCAMP, 1988). If an estimated 1.75 million systems discharge 45 gallons per day (gpd) per person, and we assume three persons per system, then 86 billion gallons of septage per year is created and potentially 51.6 billion gallons of partially treated sewage are discharging annually in New England.

Examination of the 1988 Water Quality Reports for the New England states gives some indication in a few states of the possible extent of septic problems. Failing septic systems are noted as a major contributor to the nonattainment status of 27 of 32 drainage basins in Massachusetts and as a minor contributor to two other basins. In New Hampshire it has been noted that on-site wastewater systems had moderate to minor impacts on 92.2 miles of rivers, and in Maine approximately 31.3 percent of ground-water nonattainment areas resulting from nonpoint sources (or 91.3 square miles) are estimated to be due to septic systems.

The road de-icing salts category includes the application and storage of de-icing salts and sand/salt mixtures. Table 19-5 displays various types of road salt statistics available from the states and other sources. Using the tonnage per year figures for some of the states, an estimation can be made that 750,000 tons of salt are applied annually to major New England roads. In Maine it is estimated that a total of 17.9 percent of ground-water nonattainment areas resulting from nonpoint sources are due to road salt. Plumes from 700 uncovered storage piles account for 3.8 percent (or 10.9 square miles) and road salt applications account for 14.1 percent (or 41 square miles). A Rhode Island study (FHA, 1981) showed high concentrations of sodium and chloride thousands of feet downgradient from salt storage piles.

For the Class V underground injection wells, a diverse array of source types and an alarmingly large potential for unrecognized ground-water contamination are included in the category. By definition, a Class V well injects nonhazardous fluids into or above underground sources of drinking water. However, many of these wells, by nature of design and function, directly discharge an array of toxic pollution sources above or into ground water. If these wells are injecting hazardous substances they should be classified as Class IV wells, which have been banned nationally, and thus would have to be shut down or have their discharge modified and regulated by permit. Table 19-6 is the May 1987 Class V well inventory for New England as reported to Congress. The scatter and various weights of numbers in Table 19-6 suggest an incomplete inventory of Class V wells in New England and illustrate the variation in targeting priorities among the states with respect to the inventory of specific types of wells. More recent figures available from a EPA Federal Underground Injection Control Reporting System (FURS) database report, dated February 5, 1988, indicate a total of 449 wells, and, according to the states' files on data not yet entered into FURS, there are 729 Class V wells in New England. The discrepancies are partially due to a new system of well codes for reporting inventories and are being resolved. Table 19-7 lists what are classified as Class V wells under the new reporting system. Note that wells involving disposal of septage are for systems serving 20 persons or more.

Table 19-5

Annual Road De-Icing Salts

State	Tons Per Year	Two-Lane Miles of Road	State and/or Local Road	Pounds per Lane Mile Per Storm Event	Storage Capacity	Covered ?
Connecticut	--	--	--	160	--	--
Maine	55,000	22,000	both	160	700 piles	no
Massachusetts	259,367	--	state	300	--	--
New Hampshire	139,434	8,571	--	250	123,305 tons	yes
Rhode Island	--	--	--	150-300	91 piles	--
Vermont	90,000	3,000	state	250	--	--

Table 19-6

New England Class V Well Inventory

(Note: refer to Table 19-7 for well codes)

State	Total	5D2	5D3	5D4	5A7	5W11	5W12	5A19	5W20	5X28	5R21	5X26
CT	84	3	--	--	12	62	--	--	6	1	--	--
ME	15	--	--	--	--	--	--	--	15	--	--	--
MA	132	19	--	--	10	27	72	3	1	--	--	--
NH	38	--	3	16	2	--	--	3	13	--	1	--
RI	80	--	--	--	--	8	--	8	59	3	--	2
VT	15	--	--	--	--	--	--	--	5	10	--	--
Totals	364	22	3	16	24	97	72	14	99	14	1	2

Source: U.S. EPA, May 1987.

Table 19-7

Types of Class V Wells

Well Code	Well Description
5W9	Untreated sewage waste disposal wells
5W10	Cesspools
5W11	Septic systems (undifferentiated disposal method)
5W31	Septic systems (well disposal method)
5W32	Septic systems (drainfield disposal method)
5W12	Domestic WWTP effluent disposal wells
5A19	Cooling water return flow wells
5W20	Industrial process water and waste disposal wells
5X28	Automobile service station disposal wells
5F1	Agricultural drainage wells
5D2	Storm water drainage wells
5D3	Improved sinkholes
5D4	Industrial drainage wells
5G30	Special drainage wells
5A5	Electric power reinjection wells
5A6	Direct heat reinjection wells
5A7	Heat pump/air conditioning return flow wells
5A8	Ground-water aquaculture return flow wells
5R21	Aquifer recharge wells
5B22	Saline water intrusion barrier wells
5S23	Subsidence control wells
5X13	Mining, sands, or other backfill wells
5X14	Solution mining wells
5X15	In-situ fossil fuel recovery wells
5X16	Spent-brine return flow wells
5X17	Air scrubber waste disposal wells
5X18	Water softener regeneration brine disposal wells
5N24	Radioactive waste disposal wells
5X25	Experimental technology wells
5X26	Aquifer remediation wells
5X29	Abandoned drinking water wells
5X27	Other wells

Potential sources of agricultural pesticide and fertilizer contamination include land application, disposal, spills and leaks by manufacturers and formulators, dealers, and industrial, agricultural, and domestic users. For this assessment, only regional agricultural applications of pesticides were estimated.

A study extrapolating applications per year of 12 pesticides, based on 1979 and 1982 national data, indicates that 2.04 million pounds per year are applied in New England, or about 1,020 tons per year. This estimate does not include homeowner use, usage in forests, rights of way, golf courses, and other nonagricultural uses (RFF, 1985). The pesticides that generally have a higher potential to leach into ground water are those with soil half-lives greater than two to three weeks (USEPA, Feb. 1988). Probably less than 1 percent of the estimated 1,020 tons per year of agriculturally applied pesticides leach into ground water.

Fertilizers are a source of nitrates and leach into ground water much more readily than pesticides. In Rhode Island, a total of 22,849 tons of fertilizer were used last year, and only 9 percent of that was for agricultural use, the rest being for lawn and golf course applications.

The only exposure pathway considered for all sources was ingestion of contaminated ground water via private drinking-water supplies. Contamination of public drinking-water supplies was presumed to be incorporated in the Drinking Water problem area (#12).

Estimates of Population Exposures

Septic Tanks and Cesspools

Examination of Tables 19-1 and 19-2 indicates that a large range of health effects could result from excessive concentrations of any one of, or a combination of, the contaminants in these lists. In order to evaluate the risk of nitrate contamination, we would need concentration data and an estimate of the infant population using private well water. We have chosen to evaluate microorganism contamination for the non-carcinogenic risk and only one toxic chemical for the carcinogenic risk of septic tanks and cesspools.

An EPA report on septic tanks (June 1987) presented data showing that nationally, between 1971 and 1979, 63 percent of cases of illness caused by untreated and contaminated ground water were caused by the overflow of, or leachate from, septic tanks or cesspools. The report further states that "septic tanks represent a significant threat not only to preserving the potability of ground water, but to human health." The report cites several case studies for which viruses and bacteria were found in ground water from the point of discharge to as far as 175 feet away in areas with shallow water tables. The report concludes that there is potential for microorganisms to travel great distances and certainly to reach private wells within 100 to 200 feet from the point of discharge. Several other case studies of disease outbreaks attributable to septic tanks note acute gastroenteritis and hepatitis A as frequently occurring health endpoints. These diseases were also noted as the most common in Rhode Island's

1988 Water Quality Assessment report. A survey of private well contamination in Massachusetts (Commonwealth of MA, 1988) states that although "exact figures on sodium, bacteria, and nitrate levels were not obtained as a result of this survey, it is likely that either sodium, bacteria, or nitrate has affected a private well in every municipality in the Commonwealth." The true extent of disease due to bacterial or viral contamination of ground water is unknown for several reasons. Private wells are not regularly monitored; coliform bacteria levels may not correspond directly to levels of other microorganisms, especially some small viruses that can travel farther; and there is an underreporting of diseases or a lack of awareness that contaminated ground water is at cause.

Several documents were referred to regarding the toxic chemical pollution potential of septic tanks and cesspools. Briefly, if toxic chemicals are present in domestic wastewater they generally will find their way into the ground water because septic systems are not designed to remove these chemicals. Also, most septic tank cleaners are up to 100 percent solvents by weight and tend to do more damage than good to the septic tank by killing the naturally occurring microorganisms that help treat the septage. A Rhode Island Individual Sewage Disposal Systems (ISDS) Task Force report (ISDS, 1986) references a case study where a sampling of septic tank effluent revealed 40 to 50 compounds at >1 ppb, five of which were identified as EPA priority pollutants. Another referenced study found 1 to 10 ppb concentrations of organic chemicals in the ground water near septic systems. For this evaluation we assumed an average exposure concentration for methylene chloride of 1 ppb. However, it should be kept in mind that if a septic system is malfunctioning such that a well is being contaminated, then it is most likely that a variety of organic and inorganic chemicals will be present in varying concentrations.

The population at risk from ground water contaminated by septic tanks and cesspools is estimated at 1.2 million people. Approximately 2 million people use private wells in the New England region. If there are an estimated 1.75 million septic systems serving two to three people on average, then septic systems serve 3.5 million to 5 million people, many more than private wells do. We cannot assume that every household on a septic system is on a private well, but we can assume that every household with a private well is on a septic system. This leads to a predicament: how do we know if the estimated number of malfunctioning tanks (60 percent of the total) serves households on private wells? There is no easy way to determine this; thus we estimated that 60 percent of the households served by a private well have a malfunctioning septic system, which would place 1.2 million people at risk of drinking contaminated ground water.

Road De-Icing Salts

Private wells are being contaminated by salt storage piles and road salt applications, although most states are now covering their storage piles. Limited research reveals well-contamination and well-closing statistics that are not conducive to a quantitative risk assessment; that is, average concentration data are needed, rather than numbers of drinking water violations, to conduct a quantitative risk assessment. The following discussion of sodium and chloride contamination of private wells qualitatively indicates the extent of the problem. The statistics are based on a sodium standard of 20 mg/l and a chloride standard of 250 mg/l. (Although chloride is not a health problem, it is used as a contamination indicator by those states with naturally occurring high sodium levels.)

Vermont estimates that seven wells close each year due to excessive sodium and chloride levels. New Hampshire replaces an average of 36 wells per year due to excessive chloride levels. Both states note that it is primarily shallow, dug, or driven wells that are affected. In Maine, 66 private wells, mostly drilled wells, were contaminated by chloride recently (this is probably not an annual number). Connecticut notes that 18 wells were contaminated by sodium between 1986-1987, bringing a recent total to 61 wells. Rhode Island had private well contamination surrounding eight of their storage sites, with sodium levels as high as 300 mg/l.

The population at risk from road salts would be that portion of the approximately 2 million private well users who are on a restricted salt diet and whose well has been contaminated by sodium levels greater than 20 mg/l.

Class V Underground Injection Wells

We did not cite any data regarding the contamination of private wells in New England specifically due to Class V wells. It is presumed that this source of contamination may affect those wells contaminated by an "unknown source." Once the states begin to fully inventory and locate Class V wells, especially with regard to their proximity to ground-water wells, private well testing may better identify this source area's contamination potential.

The population at risk may be any portion of the approximately 2 million private ground-water well users in Region I, although the actual total number depends on the location of the wells relative to points of ground-water use.

Agricultural Pesticides and Fertilizers

Maine, Massachusetts, Rhode Island, and Connecticut have the highest potential of the New England states for ground-water contamination by pesticides. It is thought that, in general, much less than 1 percent of the agriculturally applied pesticides leach into ground water, although this would probably vary considerably depending on soil and other conditions. Since pesticides are very expensive to test for, citizens are reluctant to voluntarily test their wells, and thus the extent of contamination is unknown. Private well testing by the states, when funding is available, is usually targeted for high risk agricultural areas.

Rhode Island has texted extensively for aldicarb in the southern area of the state. Total aldicarb was positive in 457 of 2,952 samples (15 percent) taken from 1,136 wells, with 145 samples (or 5 percent) above the health advisory of 42 ppb. Examination of preliminary results (not yet published) shows a mean concentration of 6 ppb for all positive samples and a mean concentration of 52 ppb for the 5 percent above the 42 ppb health advisory level.

Connecticut noted 134 wells as being contaminated by pesticides between 1986-1987, bringing the total of well contamination incidents to 417. Massachusetts found 77 wells in eight communities contaminated by chemical pesticides. A private well survey, testing wells in 27 Massachusetts communities for eight pesticides, found 61 wells above state interim guidelines with a total of 155 wells showing some detectable concentration. Of the 61 wells over the standard, 24 were for EDB, 33 for 1,2-D, 22 for aldicarb, 1 for carbofuran, 1 for alachlor, and 2 for dinoseb. The three-year study was conducted from 1983 to 1986. A 1987 survey found six wells in one community over the standards: one for alachlor, four for atrazine, and one for metolachlor.

The population at risk from pesticide-contaminated ground water would be that portion of the estimated 2 million private ground-water well users living in the vicinity of agricultural areas.

Risk Characterization

It was very difficult to determine whether a quantitative risk characterization of this problem area was feasible. Initially, the work group felt that an improvement could be made on the results of the national comparative risk project, especially where the only estimate of carcinogenic potential for a list of sources even longer than that considered here was based on one septic tank contaminant. However, a lack of data left us in the same predicament. The main risk characterization attempt was to improve the national project's qualitative discussion of the potential sources covered and to determine the possible extent of the problems in New England. Since the exposed population considered was some portion of the estimated 2 million people using domestic ground-water wells, and water quality data for these wells are incomplete, a qualitative assessment was generally all the work group could do.

The only attempts to quantify some of the risk for this problem area concern septic tanks for both the non-carcinogenic and the carcinogenic potential. We felt that the risk of disease by bacterial and viral contamination facing an estimated 60 percent of the private well user population would drive the non-carcinogenic risk. Other non-carcinogenic risk is posed by the organic and inorganic chemicals commonly occurring in domestic wastewater, by sodium from road de-icing salts, by the potential contaminants from Class V underground injection wells, and by agricultural chemicals, although the exposed population for these sources is probably much lower than that exposed to microorganism contamination.

The carcinogenic potential of this category was nearly impossible to estimate. An attempt was made to quantify risks from methylene chloride, also known as dichloromethane, one of many possible toxic organic chemicals commonly found in septic tank leachate. The estimated individual risk posed by methylene chloride is 2E-7, a lifetime population cancer risk of less than one cancer case for an estimated 1.2 million people and an annual population cancer risk of much less than one cancer case (based on a 70-year lifespan). The estimate was based on an average concentration of 1 ppb, which was the range noted in a few case studies, and not on actual, regional concentration data. If more concentration data were available on the occurrence in ground water of the organic chemicals from all of the sources being assessed in this problem area, the cancer case number would most likely be higher.

The uncertainty in the ranking is high, based on the qualitative nature of the analysis compared to other problem areas in the risk evaluation project. The percentage of the problem area covered is low.

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20. & 21. Pesticide Residues & Pesticide Application

Problem Area Definition

These two problem areas address the health risk associated with pesticide residues on food and pesticide application. Analysis of pesticide residues on food includes consideration of residues on foods imported into the region as well as residues on food grown in the region. Analysis of risks from pesticide application includes risk to professional applicators, home applicators, and bystanders. This analysis also covers improper application and accidental exposure.

Summary/Abstract

The risks from pesticide residues and applications are primarily calculated by apportioning the national risk to Region I. For pesticide residues, the national risk (6,000 cancer cases) is apportioned based on the regional population, resulting in an estimated 317 annual cancer cases in New England. The national cancer risk from pesticide application (100 cases) is apportioned according to use of pesticides in the region. Using this method, less than one annual cancer case is associated with application in Region I. Non-cancer effects are associated only with the application of pesticides. Using hospital admission figures and a pesticide poisoning incidence rate, approximately 27 applicators and farm workers are admitted to hospitals for pesticide-related health effects. Impacts on home applicators and bystanders are not assessed.

Toxicity Assessment

Specific pesticides are not addressed in this analysis. Risks are estimated based on the national risk estimates calculated in *Unfinished Business*. For effects from pesticide application, the acute risks are based on hospital admissions. These admissions are assumed to be related to respiratory problems.

Exposure Assessment

More than 200 pesticide chemicals are potentially oncogenic. Specific concentrations of these pesticides are not estimated. The potential pathways of exposure include ingestion of pesticide residues on food and direct inhalation of and contact with pesticides being applied. The total regional population is assumed to be exposed to pesticide residues on food. No calculation of total population exposed is made for pesticide application.

Risk Characterization

The cancer risk associated with pesticide residues is calculated by apportioning the national risk (6,000 cases annually) from *Unfinished Business* to Region I, based on the region's percentage of the U.S. population. This results in an annual incidence of 317 cases for the total New England population. We did not estimate non-cancer risk associated with pesticide residues on food.

The cancer risk from pesticide application is determined by apportioning the national estimate (100 cases annually) to the region based on annual pesticide use. The result is less than one annual cancer case.

For pesticide applications, the acute risks are calculated by multiplying the number of hospital admissions in Region I (1.8 million) by a pesticide poisoning incidence rate. The pesticide poisoning rate used from EPA's *National Study of Hospital Admitted Pesticide Poisonings* is 0.6 per 100,000 people for applicators, and 0.9 per 100,000 people for farm workers. This results in an annual incidence of 11 applicator and 16 farm worker hospitalizations. The impacts on home applicators and bystanders are not assessed.

The uncertainty associated with the risks from pesticide residues and application is high. The calculation of cancer cases from pesticide residues assumes that estimates from *Unfinished Business* are accurate and that exposure across the region is equivalent. The calculation of risk associated with pesticide application assumes that all pesticides are of equal toxicity. The use of hospital data in this case may bias the results downward because it is generally believed that a significant percentage of acute poisonings go unreported.

22. Lead

Problem Area Definition

This category considers risks from exposure to lead in soil, drinking water, and air.

Summary/Abstract

The lead problem area ranks in the highest category for public health risk. The driving health endpoint is learning disabilities in young children. The basis for this analysis is the prevalence of confirmed cases of lead poisoning in young children as reported by the six New England states. Recent data noted 1,951 confirmed cases of lead poisoning in New England, and approximately 10,000 additional cases would be anticipated if all children less than six years of age were screened for lead. Lead is not considered a carcinogen. The major exposure routes for lead in New England are dust, paint and soil. Minor exposure routes include drinking water and air.

Toxicity Assessment

Lead metal and its compounds are the materials of concern in this problem area. The compounds include acidic lead (lead acetate), isocyanic acid (methyl isocyanate), lead phosphate (phosphoric acid), lead stearate, and lead sulfide. For simplicity, the term "lead" will be used to cover lead metal and the lead compounds.

Health Effects of Concern

Non-Cancer Health Effects

There is no convincing evidence that lead has any beneficial biological effect in humans. (Expert Committee on Trace Metal Essentiality, 1983; and included in the Lead Criteria document, 1986.)

Elevated blood-lead levels have been linked to a wide range of health effects, which are of particular concern in young children. These effects range from relatively subtle changes in biochemical measurements at 10 ug/dl (micrograms/deciliter) and below to severe retardation and even death at very high levels (80 to 100 ug/dl). Lead can interfere with blood-forming processes, vitamin D metabolism, kidney function, neurological processes, and reproductive functions in both males and females. In addition, the negative impact of lead on cognitive performance (as measured by IQ tests, performance in schools, and other means) is generally accepted at moderate-to-high blood-levels (30 to 40 ug/dl and above), and several studies also provide evidence for possible attentional and IQ deficits at levels as low as 10 to 15 ug/dl. Changes in electroencephalogram readings, as another example, have also been observed at low levels. For many subtle effects, the data may represent the limits of detectability of biochemical and other changes and not necessarily actual thresholds for effects (Ronnie Levin, *Reducing Lead in Drinking Water: A Benefit Analysis*, OPPE, USEPA, 1986).

This analysis uses learning disabilities as the non-cancer health endpoint of concern. This endpoint is the health problem most frequently encountered in the most vulnerable population, children less than six years of age. However, it is important to understand that the evaluation of learning disabilities in young children will tend to understate the overall health risks posed by lead. Severe lead intoxication can be fatal, for example. While this result is no longer common (lead poisoning was a leading cause of death for American children in the early part of this century), the risk remains. As indicated above, lead is associated with a range of serious health problems in other populations, particularly women of childbearing age and middle-aged males. Unfortunately data are insufficient on lead exposure or incidence of lead poisoning for these groups. Since this analysis does not consider the rare cases of severe lead intoxication or the effects of lead on all segments of society, it represents a lower bound on the public health risk posed by lead in New England.

Cancer Health Effects

Lead is classified as a Group B 2 carcinogen by EPA, indicating that there is some evidence of carcinogenicity from animal studies. However, several epidemiology studies have not shown an association between lead exposure and cancer incidence. This analysis does not consider cancer risks posed by lead.

Exposure Assessment

The data used in this analysis are prevalence data on lead poisoning in children in New England.

The Center for Disease Control (CDC) defines lead poisoning as 25 micrograms per deciliter (ug/dl) of lead in blood with free erythrocyte protoporphyrin (FEP) greater than 35 ug/dl. FEP is used as an indicator of potential lead poisoning.

Most New England communities conduct some lead screening for children less than six years of age. Screening programs take a capillary blood sample from a child and analyze the sample for FEP. If the FEP is greater than 35 ug/dl, the parents are contacted and a venous blood sample is taken from the child. This sample is analyzed for lead. If this blood lead is greater than 25 ug/dl, the child is counted as a confirmed case of lead poisoning.

State health departments maintain the results of screening programs but there is no common format for recording or reporting this information. All six New England state health departments were contacted for this analysis. Massachusetts, Maine, New Hampshire, and Rhode Island reported the percentage of children screened and the number of confirmed cases of lead poisoning. Connecticut reported the percentage screened and the percentage of children presumed at risk. Vermont reported that both targeted and broad-based screening efforts in recent years have identified only one confirmed case of lead poisoning.

The following table summarizes the information received from state health departments.

State	Number of Children Screened		Cases
Connecticut	-	(≤ 12%)	(6,060 potential)
Maine	4,151	(4%)	249
Massachusetts	165,852	(39%)	1,235
New Hampshire	7,150	(10%)	39
Rhode Island	17,455	(24%)	427
Vermont	Screening data not reported		1

Pathways of Exposure

This analysis does not isolate any particular exposure route or set of exposure routes for lead. Rather it relies on data on the prevalence of lead poisoning, which reflects exposure from all pathways.

Lead is a ubiquitous environmental contaminant. It is found in air, water, and soil as well as in plumbing, housepaint, food, and dust. This section discusses the exposure routes of lead and the risks they present.

As stated above, lead is found in every medium throughout the environment. Virtually all lead contamination is the result of human activity. Furthermore, lead moves easily from one medium to another. The major contributors to lead poisoning in New England are lead paint, lead soil, and lead dust. Minor but still significant exposure routes are lead in air and lead in drinking water.

Lead paint is common in homes constructed before 1950. As many as 85 percent of these homes may contain lead paint. The age and condition of the housing stock are predictors of lead poisoning. Lead poisoning cases are most often found in older inner city neighborhoods. However, cases are also routinely reported in suburban and rural communities. Children are at risk wherever they are exposed to lead.

Cases of acute lead poisoning (greater than 40 ug/dl) are usually the result of ingestion of lead paint. These cases are less common than in the past.

The most common cases are that of blood lead between 25 ug/dl and 40 ug/dl. Recent research indicates that inhalation or ingestion of lead-contaminated dust is the critical exposure pathway for these cases. The relationships between paint, soil, and dust are not well understood. Researchers believe that soil is contaminated with lead from deteriorating interior and exterior lead paint, from deposition of airborne lead, and from lead in water. All of these sources contribute to the contamination of dust. It is important to note that lead does not leach through soil. Rather, it remains where it was deposited. As a result, surface soil contains the accumulation of years of lead pollution from gasoline, old paint, etc. The soil acts as a reservoir of lead contamination.

CDC's most recent statement on lead poisoning (1985) asserts that soil contaminated at 500 to 1,000 parts per million (ppm) lead or more is associated with significant increased risk for lead poisoning in children. Data on soil contamination in residential areas is scarce. Sampling conducted in Boston in October 1987 identified average surface soil levels greater than 1,900 ppm lead.

The reduction of lead in gasoline has led to a dramatic reduction in airborne lead. However, most of the airborne lead from past years is probably now deposited as lead in soil.

Lead in drinking water remains a problem. Recent tests in Boston revealed that more than half of the city schools have unsafe levels of lead in their drinking water, primarily as a result of slightly acidic water interacting with old lead pipes and lead solder in plumbing fixtures. The lead levels in drinking water are unlikely to cause lead poisoning in the absence of other sources, but they do contribute to the overall body burden of lead. This in turn increases the risk of lead poisoning from other sources.

Lead in drinking water may be a major source of concern for adults. Adults tend to drink more water than children and because of behavior patterns are likely to be at lower risk from exposure to soil, dust, and paint.

Population at Risk

The population at risk is all children under six years of age.

State	Children Under Six Years
Connecticut	202,000
Maine	103,770
Massachusetts	430,143
New Hampshire	71,084
Rhode Island	72,842
Vermont	47,459
Total	927,298

Risk Characterization

An estimate of lead poisoning cases in New England was determined by extrapolating from existing screening and prevalence data to project the number of cases if all children under six years were screened.

The first step in the calculation was to total all of the reported confirmed cases of lead poisoning, giving a total of 1,951.

The next step was to extrapolate from the known cases to the number of expected cases if screening programs covered 100 percent of the children under six. The analysis used a weighted average to determine expected cases in Maine and Rhode Island because the high rates in these states seemed to reflect the screening of the most vulnerable children. Vermont was not used in the calculation of the weighted average. The rate (#cases/#screened) for each state was multiplied by the number of children screened in each state as a percentage of the total number of children screened in New England. The resulting weighted fractions for each state were then summed to get a weighted rate of 1.2 percent. The weighted rate was used to determine expected cases in Maine, Rhode Island, and Vermont. For Massachusetts and New Hampshire, expected cases were determined by using the rate for that state. For Connecticut, the 3 percent rate was used.

The final step was to combine reported cases with expected cases.

Connecticut:	6,060	(3% of children under six)
Maine:	1,444	(249 reported + 1,195 expected)
Massachusetts:	3,191	(1,235 reported + 1,956 expected)
New Hampshire:	391	(39 reported + 352 expected)
Rhode Island:	1,092	(427 reported + 665 expected)
Vermont:	571	(1 reported + 570 expected)
Total:	12,749	(1,951 reported + 10,798 expected)

Projected Overall New England Rate: 1.37 percent

These numbers are not meant to reflect the precise number of cases that exist in New England or in any individual state. Rather, they reflect the approximate magnitude of health risk to children from lead poisoning.

When this calculation is placed in the format of the Comparative Risk Project, it produces the following results.

Population Score:	3
Health Endpoint Score:	3
Ratio Score:	4
Total Score	10

Using a different methodology for calculating the expected cases of lead poisoning would not change the scoring results. The only factor that is not fixed is the ratio score. Using only the 1,951 confirmed cases in New England, the rate is 10-2 (1,951 cases/194,608 children screened). This value still results in a ratio score of 4.

One test of the validity of these results is comparison with other studies. The data from the Second National Health and Nutrition Examination Survey (NHANES II) (1976-1980) indicate that 4 percent of American children less than five years old have blood-lead levels at or above 30 ug/dl. This implies that the rate developed in this analysis, 1.37 percent, is likely to be a lower bound. The actual rate, therefore, may be higher.

The uncertainty in this analysis is low. The data collected are reliable and the calculations are simple. The estimated number of lead poisoning cases--12,749--is believed to be the correct order magnitude.

Percentage of Problem Covered

The percentage of problem covered for lead exposure in young children is high, and this is widely accepted as the most vulnerable population. As indicated above, there are other populations at risk from lead, including women of childbearing age and middle-aged men. However, since exposure data for these groups are limited and lead is ranked in the highest residual public health risk category without including an analysis of these groups, further analysis was not conducted.

23. Asbestos

Problem Area Definition

Asbestos has become an issue of great environmental concern because of the epidemiological evidence showing a strong correlation between cumulative asbestos exposures and deaths. Asbestos is associated with such lethal diseases as lung cancer and mesothelioma (a relatively rare cancer of the thin membranes lining the chest and the abdomen). Asbestos has also been linked to gastrointestinal (GI) cancers and cancers at other anatomical sites, including the larynx, kidney, and ovary.

Asbestos can be released to the ambient air at numerous stages in its production and use. There are three general types of asbestos exposure:

- Occupational (e.g., worker exposures at facilities involved in mining and milling, fiber processing into products, installation of products, building maintenance)
- Non-occupational (e.g., exposures resulting from time spent in asbestos-containing buildings, such as schools, office buildings, and residences)
- Ambient (e.g., general background levels resulting from construction and demolition of buildings containing asbestos, asbestos removal; in some areas, elevated background levels may be found because of the existence of either large manufacturing operations using asbestos or asbestos mining and milling facilities)

This evaluation developed rough approximations of the risks to human health resulting from the latter two exposure scenarios.

Summary/Abstract

Based on the results of a risk assessment completed by EPA's Office of Health and Environmental Assessment, we developed screening-quality estimates of individual risk and annual incidence for three cancer types (lung cancer, mesothelioma, and GI cancer) and three exposure scenarios (rural ambient air--0.07 ng/m^3, urban ambient air--3 ng/m^3, and non-occupational/indoor air--80 ng/m^3). The risk assessment considered only exposures through inhalation. Because of data limitations on the number of individuals exposed to asbestos in Region I, we assumed conservatively that the total Region I population was exposed to asbestos in the ambient air--subdivided by residence in either rural or urban areas--and in non-occupational settings. This approach allowed us to develop a range of incidence projections that we feel bounds the problem for screening purposes.

The estimated lifetime individual cancer risks for non-occupational exposures ranged from 1E-5 (GI cancer) to 1E-4 (mesothelioma and lung cancer). The estimated lifetime individual cancer risks for urban and rural ambient exposures were, respectively, one and two orders of magnitude smaller. The analysis predicted approximately five annual cases of cancer from ambient air exposures. The urban exposures accounted for almost all of the estimated incidence (approximately 99 percent). The estimated annual cancer incidence associated with non-occupational exposures was significantly higher, contributing roughly 178 annual cancer cases.

Toxicity Assessment

Asbestos has a strong association with lung cancer and mesothelioma. It has also been linked to GI cancers and cancers at the larynx, kidney, and ovary; however, the degree of risk and the strength of the evidence supporting these diseases are less than for lung cancer or mesothelioma. The International Agency for Research on Cancer lists asbestos as a Group 1 carcinogen; EPA's guidelines characterize asbestos as a Group A human carcinogen.

To quantify the risks associated with lung cancer and mesothelioma, we relied on the risk calculations presented in the EPA document *Airborne Asbestos Health Assessment Update,* EPA/600/8-84/003F, June 1986. The available data suggest that the excess risk of lung cancer from asbestos is proportional to the cumulative exposure (the duration times the intensity) and the underlying risk in the absence of asbestos exposure. The risk of death from mesothelioma is approximately proportional to the cumulative exposure to asbestos and increases exponentially with time since the initial exposure. This analysis assumed conservatively a lifetime exposure starting at birth; it did not consider smoking habits, which can increase the risks. Thus, the analysis used the following approximate cancer unit risk factors:

- 9.3E-6 (f/ml)-1 for mesothelioma (women)
- 6.3E-6 (f/ml)-1 for mesothelioma (men)
- 1.7E-6 (f/ml)-1 for lung cancer (women)
- 5.7E-6 (f/ml)-1 for lung cancer (men)

The information available is insufficient to quantify the dose-response relationships for other asbestos cancers. However, excess GI cancer mortality is approximately 10 to 30 percent of the risk attributable to lung cancer. We calculated GI cancer risks using a midpoint of 20 percent.

Asbestos is also associated with non-cancer effects. Exposure to asbestos may increase the risk of asbestosis, a chronic lung ailment resulting in shortness of breath, permanent lung damage, and an increased risk of lung infections. However, there was insufficient information to estimate the occurrence of asbestosis in this preliminary risk screen.

Exposure Assessment

Asbestos can be released at numerous points in its production and use. This analysis considered only ambient air and non-occupational exposures. Because these points of exposure are ubiquitous, we relied on available monitoring data to characterize average concentrations found in each exposure setting. Table 23-1 presents the mean measured concentration values from studies (both ambient and indoor) deemed by EPA to have a reasonable number of sampling episodes.

Ambient air exposures were divided into two categories: rural and urban. An average ambient air concentration of 3 ng/m^3 was used to predict cancer risks in urban environments. The city monitoring data in Table 23-1 generally support this value, with the exception of the findings for Paris, France. Also, EPA's 1985 analysis of hazardous air pollutants used an average asbestos ambient level of 3 ng/m^3 in its analysis of cancer risks.[1]

Because large fractions of the population in three Region I states (Vermont, New Hampshire, and Maine) reside in rural areas, it was important to consider asbestos levels found in more remote areas. Atmospheric sampling programs conducted in rural areas in the United States and Germany detected asbestos fiber levels between 0.01 and 0.12 ng/m^3 (corresponding to roughly 3E-7 f/ml and 4E-6 f/ml, respectively).[2] A midpoint of 0.07 ng/m^3 was used as the representative concentration for rural areas.

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[1] U.S. EPA, Office of Policy Analysis, *An Analysis of Carcinogenic Risks for Selected Pollutants Nationwide*, May 1985.

[2] *Federal Register* 40 CFR Part 763, "Asbestos; Proposed Mining and Import Restrictions and Proposed Manufacturing Importation and Processing Prohibitions," January 29, 1986.

Table 23-1

Summary of Environmental Asbestos Sampling

Sample Set	Collection Period	Number of Samples	Mean Concentration ng/m^3
Quarterly composites of 5 to 7 24-hour U.S. samples (Nicholson, 1971; Nicholson and Pundsack, 1973)	1969-1970	187	3.3C[a]
Quarterly composite of 5 to 7 24-hour U.S. samples (U.S. EPA, 1974)	1969-1970	127	3.4C
5-day samples of Paris, France (Sebastien et al., 1980)	1974-1975	161	0.96C
6- to 8-hour samples of New York City (Nicholson et al., 1971)	1969	22	16C
5-day 7-hour control samples for U.S. school study (Constant et al., 1983)	1980-1981	31	6.5 (6C, 0.5A[b])
16-hour samples of 5 U.S. cities (U.S. EPA, 1974)	1974	34	13C
New Jersey schools with damaged asbestos surfacing materials in pupil-use areas (Nicholson et al., 1978)	1977	27	217C
U.S. school rooms/areas with asbestos surfacing materials (Constant, 1983)	1980-1981	54	183 (179C, 4A)
U.S. school rooms/areas with asbestos surfacing materials (Constant, 1983)	1980-1981	31	61 (53C, 8A)
Buildings with asbestos materials in Paris, France (Sebastien et al., 1980)	1976-1977	135	35 (25C, 10A)
U.S. buildings with friable asbestos in plenums or as surfacing materials (Nicholson et al., 1975; Nicholson et al., 1976)	1974	54	48C
U.S. buildings with cementitious asbestos material in plenums or as surfacing materials (Nicholson et al., 1975, 1976)	1974	28	15C
Ontario buildings with asbestos insulation (Ontario Royal Commission, 1984)	1982	63	2.1

[a] C = Chrysotile.
[b] A = Amphibole.

An average asbestos concentration level of 80 ng/m^3 was used to characterize non-occupational exposures. This value was derived by averaging the monitoring results presented in Table 23-1 for schools and public buildings.

As a cautionary note, it is important to mention that the measured asbestos levels summarized in Table 23-1 were taken during a period of time that may not adequately reflect current conditions. Given the recent regulatory activity and the increased public awareness of the risks posed by asbestos, current levels could be lower, thus resulting in lower risk estimates. The risk screen may also result in overestimates of risk because data limitations made it necessary to assume conservatively that all asbestos exposures started at birth and continued for a lifetime. The individual risk results presented can be easily adjusted to reflect alternate concentration levels should more recent data become available.

The primary exposure pathway for asbestos is inhalation. Some studies have also suggested the possibility of adverse human health effects through ingestion of asbestos fibers, but this route of exposure is still under investigation. To date, there is no evidence of disease incidence resulting from dermal exposures.

Because of data limitations on the population exposed to asbestos, we assumed conservatively that the total Region I population was exposed to the range of asbestos concentration levels (ambient and non-occupational) over the course of a lifetime, starting at birth. This approach allowed us to develop a range of incidence projections that we feel bounds the problem for screening purposes. U.S. census data were used to identify the total population of men and women residing in Region I. Census data were also used to determine the proportion of men and women residing in metropolitan and nonmetropolitan areas for each Region I state to complete the excess cancer incidence calculations for ambient air exposures. The population data used in this analysis are summarized in Attachment 23-B.

Risk Characterization

The lifetime individual cancer risks for non-occupational exposures could range as high as 1E-4, whereas ambient exposures are two to four orders of magnitude lower. Ambient urban exposures are associated with total lifetime individual cancer risks of approximately 1E-5; the total lifetime individual cancer risk for remote rural areas is approximately 1E-7. Table 23-2 summarizes the estimated lifetime individual cancer risk estimates for each concentration scenario and health effect category.

Table 23-2

Estimated Lifetime Individual Cancer Risks for Asbestos

Health Effect	Ambient Rural Exposures[1] (0.07 ng/m^3)	Ambient Urban Exposures[2] (3 ng/m^3)	Non-Occupational Exposures (80 ng/m^3)
Mesothelioma:			
-- Males	4.4E-07	1.9E-05	5.1E-04
-- Females	6.5E-07	2.8E-05	7.5E-04
Lung Cancer:			
-- Males	4.0E-07	1.7E-05	4.5E-04
-- Females	1.2E-07	5.0E-06	1.4E-04
GI Cancers:			
-- Males	7.9E-08	3.4E-06	9.1E-05
-- Females	2.3E-08	1.0E-06	2.9E-05

Note: These screening-quality risk results are for use only in establishing a relative ranking of environmental issues for further study.

[1] Based on information presented in 40 CFR Part 763, January 29, 1986, remote rural concentrations were found to range between 0.01 ng/m^3 and 0.12 ng/m^3. For this analysis, a midpoint of 0.07 ng/m^3 was used. Risk results were linearly extrapolated from the EPA risk results.

[2] Source: U.S. EPA, Office of Health and Environmental Assessment, *Airborne Asbestos Health Assessment Update*, EPA/600/8-84/003f, June 1986, p. 165.

[3] Estimated as 20 percent of the predicted lung cancer risks.

Table 23-3 presents the predicted annual incidence of cancer associated with ambient and non-occupational exposures. As shown, total ambient exposures (rural and urban) contribute approximately five excess cancer cases annually. The incidence estimates for non-occupational exposures are more than two orders of magnitude higher, potentially reaching as high as 176 annual excess cancer cases.

To properly interpret and use the asbestos risk screen results in the risk evaluation project, it is important to understand the limitations of the risk screen methodology. These uncertainties are grouped based on whether they are expected to yield overestimates or underestimates of risk.

- Assumptions/data limitations resulting in overestimates of risk
    - *Lifetime asbestos exposures start at birth.* If better site-specific information existed on this topic, we could revise the risk estimates to reflect more realistic exposure levels.
    - *All individuals in Region I are exposed to asbestos.* This is a worst-case assumption; better site-specific information on populations exposed at each concentration level would probably result in lower risk estimates.
    - *Risk projections are based on concentration data from relatively old studies.* Collection of new information would provide better indications of current asbestos concentration levels given recent regulatory activity and increased public awareness. Also, use of site-specific monitoring data would help.
    - *Extrapolation from high-dose to low-dose exposures is necessary.* There are significant uncertainties in developing risk estimates for low concentration levels based primarily on high-level occupational exposure studies. This is a concern for all risk assessments.

- Assumptions/data limitations resulting in underestimates of risk
    - *Only exposure through inhalation was considered.* There is some concern that ingestion of asbestos could pose risks. Studies are currently being conducted to explore this topic further.
    - *Lung cancer risks for smokers are significantly higher.* EPA has generated individual cancer risk estimates with and without smoking habits. Because of limited information on smokers and their exposure levels in Region I, we based the risk screen on the individual cancer risks that did not consider smoking habits. These risk estimates fell between the individual risks calculated by EPA for smokers and nonsmokers (see Attachment 23-A).

-- *Exposure levels in some buildings could be higher than 300 ng/m^3*. For example, while the mean of the measured asbestos levels in the New Jersey schools sampled (Nicholson et al., 1978, see Table 23-1) was 217 ng/m^3, the observed asbestos levels ranged from 9 ng/m^3 to 1,950 ng/m^3.

We believe that the risk assessment methodology characterized in this paper has captured most of the problem. However, the uncertainty in the risk estimates for the ambient exposures is rated medium and the uncertainty for the non-occupational exposures is rated high.

Table 23-3

Estimated Annual Cancer Incidence for Asbestos

Health Effect	Ambient Rural Exposures[1] (0.07 ng/m^3)	Ambient Urban Exposures[2] (3 ng/m^3)	Non-occupational Exposures (80 ng/m^3)
Mesothelioma:			
-- Males	0.01	1.34	44.19
-- Females	0.01	2.12	69.92
Lung Cancer:			
-- Males	0.01	1.20	39.54
-- Females	0.00	0.38	13.23
GI Cancers:			
-- Males	0.00	0.24	7.91
-- Females	0.00	0.08	2.75
Total	0.03	5.35	177.53

Note: These screening-quality risk results are for use only in establishing a relative ranking of environmental issues for further study.

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[1] Based on information presented in 40 CFR Part 763, January 29, 1986, remote rural concentrations were found to range between 0.01 ng/m^3 and 0.12 ng/m^3. For this analysis, a midpoint of 0.07 ng/m^3 was used. Risk results were linearly extrapolated from the EPA risk results.

[2] Source: U.S. EPA, Office of Health and Environmental Assessment, *Airborne Asbestos Health Assessment Update*, EPA/600/8-84/003f, June 1986, p. 165.

[3] Estimated as 20 percent of the predicted lung cancer risks.

Attachment 23-A

Table 23-A-1

Lifetime Risks per 100,000 Persons of Death from Mesothelioma and Lung Cancer from Continuous Asbestos Exposures of 0.0001 and 0.01 f/ml According to Age and Duration of Exposure. U.S. General Population Death Rates Were Used and Smoking Habits Were Not Considered.[1]

Age at Onset of Exposure	Concentration = 0.0001 f/ml Years of Exposure					Concentration = 0.01 f/ml Years of Exposure				
	1	5	10	20	Life-time	1	5	10	20	Life-time
Mesothelioma in Females										
0	0.1	0.7	1.2	2.0	2.8	14.6	67.1	120.8	196.0	275.2
10	0.1	0.4	0.8	1.2	1.5	9.4	42.6	75.5	118.7	152.5
20	0.1	0.3	0.4	0.7	0.8	5.6	25.1	43.5	65.7	78.7
30	0.0	0.1	0.2	0.3	0.4	3.1	13.3	22.4	31.9	35.7
50	0.0	0.0	0.0	0.0	0.0	0.6	2.1	3.2	3.9	3.9
Lung Cancer in Females										
0	0.0	0.0	0.1	0.2	0.5	1.0	4.6	9.2	18.5	52.5
10	0.0	0.0	0.1	0.2	0.4	1.0	4.6	9.2	18.6	43.4
20	0.0	0.0	0.1	0.2	0.3	1.0	4.6	9.2	18.2	34.3
30	0.0	0.0	0.1	0.2	0.3	1.0	4.6	9.0	16.7	25.1
50	0.0	0.0	0.1	0.1	0.1	0.7	3.1	5.5	8.1	8.8
Mesothelioma in Males										
0	0.1	0.5	0.9	1.5	1.9	11.2	61.0	91.1	145.7	192.8
10	0.1	0.3	0.6	0.8	1.1	7.0	31.2	58.2	84.7	106.8
20	0.0	0.2	0.3	0.4	0.5	4.1	17.5	30.1	44.5	51.7
30	0.0	0.1	0.1	0.2	0.2	2.1	8.8	14.6	20.4	22.3
50	0.0	0.0	0.0	0.0	0.0	0.3	1.1	1.8	2.0	2.1
Lung Cancer in Males										
0	0.0	0.1	0.3	0.6	1.7	2.9	14.8	29.7	59.2	170.5
10	0.0	0.1	0.3	0.6	1.4	2.9	14.9	29.8	59.5	142.0
20	0.0	0.2	0.3	0.6	1.1	3.1	15.0	30.0	59.4	113.0
30	0.0	0.1	0.3	0.6	0.8	3.1	14.9	29.8	56.6	84.8
50	0.0	0.1	0.2	0.3	0.3	2.5	11.5	20.3	29.1	30.2

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[1] The 95 percent confidence limit on the risk values for lung cancer for an unstudied exposure circumstance is a factor of 10. The 95 percent confidence limit on the risk values for lung cancer on the average determined from 11 unit exposure risk studies is a factor of 2.5. The 95 percent confidence limit on the risk values for mesothelioma for an unstudied exposure circumstance is a factor of 20. The 95 percent confidence limit on the risk values for mesothelioma for a studied circumstance can be reasonably averaged as a factor of 5. The values for continuous exposure were derived by multiplying 40 hr/wk risks, obtained from occupational exposures, by 4.2 (the ratio of hours in a week to 40 hours).

Table 23-A-2

Lifetime Risks per 100,000 Females of Death from Mesothelioma and Lung Cancer from Continuous Asbestos Exposures of 0.0001 and 0.01 f/ml According to Age at First Exposure, Duration of Exposure, and Smoking[1]

	Concentration = 0.0001 f/ml Years of Exposure					Concentration = 0.01 f/ml Years of Exposure				
Age at Onset of Exposure	1	5	10	20	Life-time	1	5	10	20	Life-time
Mesothelioma in Female Smokers										
0	0.1	0.6	1.2	1.9	2.5	13.9	64.0	115.1	186.2	252.0
10	0.1	0.4	0.7	1.1	1.4	9.0	40.3	71.4	112.0	142.8
20	0.1	0.2	0.4	0.6	0.7	5.3	23.5	40.7	61.3	72.8
30	0.0	0.1	0.2	0.3	0.3	2.8	12.3	20.6	29.4	32.8
50	0.0	0.0	0.0	0.0	0.0	0.6	2.0	2.9	3.5	3.5
Lung Cancer in Female Smokers										
0	0.0	0.1	0.3	0.5	1.5	2.8	13.4	26.7	53.3	149.9
10	0.0	0.1	0.3	0.5	1.2	2.8	13.4	26.7	53.3	123.5
20	0.0	0.1	0.3	0.5	1.0	2.8	13.4	26.7	52.5	96.9
30	0.0	0.1	0.3	0.5	0.7	2.8	13.3	25.9	47.9	71.0
50	0.0	0.1	0.2	0.2	0.2	2.0	8.8	15.5	22.7	24.4
Mesothelioma in Female Nonsmokers										
0	0.1	0.7	1.2	2.0	2.7	14.8	68.2	122.8	199.4	272.2
10	0.1	0.4	0.8	1.2	1.6	9.5	43.4	81.2	121.2	155.8
20	0.1	0.3	0.4	0.7	0.8	5.7	25.6	44.4	67.2	80.6
30	0.0	0.1	0.2	0.3	0.4	3.1	13.6	23.0	32.9	36.8
50	0.0	0.0	0.0	0.0	0.0	0.6	2.2	3.4	4.1	4.1
Lung Cancer in Female Nonsmokers										
0	0.0	0.0	0.0	0.1	0.2	0.3	1.3	2.7	5.2	16.4
10	0.0	0.0	0.0	0.1	0.1	0.3	1.3	2.7	5.3	13.9
20	0.0	0.0	0.0	0.1	0.1	0.3	1.3	2.7	5.2	11.3
30	0.0	0.0	0.0	0.1	0.1	0.3	1.3	2.7	5.0	8.7
50	0.0	0.0	0.0	0.0	0.0	0.3	1.1	2.1	3.5	3.9

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[1] The 95 percent confidence limit on the risk values for lung cancer for an unstudied exposure circumstance is a factor of 10. The 95 percent confidence limit on the risk values for lung cancer on the average determined from 11 unit exposure risk studies is a factor of 2.5. The 95 percent confidence limit on the risk values for mesothelioma for an unstudied exposure circumstance is a factor of 20. The 95 percent confidence limit on the risk values for mesothelioma for a studied circumstance can be reasonably averaged as a factor of 5. The values for continuous exposure were derived by multiplying 40 hr/wk risks, obtained from occupational exposures, by 4.2 (the ratio of hours in a week to 40 hours).

Table 23-A-3

Lifetime Risks per 100,000 Males of Death from Mesothelioma and Lung Cancer from Continuous Asbestos Exposures of 0.0001 and 0.01 f/ml According to Age at First Exposure, Duration of Exposure, and Smoking[1]

Age at Onset of Exposure	Concentration = 0.0001 f/ml Years of Exposure					Concentration = 0.01 f/ml Years of Exposure				
	1	5	10	20	Lifetime	1	5	10	20	Lifetime
Mesothelioma In Male Smokers										
0	0.1	0.5	0.9	1.4	1.8	10.6	48.3	85.5	137.5	181.0
10	0.1	0.3	0.5	0.8	1.0	6.6	29.4	51.5	77.8	98.3
20	0.0	0.2	0.3	0.4	0.5	3.6	16.4	28.0	41.2	47.9
30	0.0	0.1	0.1	0.1	0.2	2.0	8.1	13.4	18.5	20.2
50	0.0	0.0	0.0	0.0	0.0	0.3	1.1	1.5	1.8	1.8
Lung Cancer In Male Smokers										
0	0.0	0.2	0.4	0.8	2.4	4.2	20.9	41.9	83.4	238.1
10	0.0	0.2	0.4	0.8	2.0	4.2	21.0	42.0	83.9	197.8
20	0.0	0.2	0.4	0.8	1.6	4.2	21.3	42.3	83.4	157.5
30	0.0	0.2	0.4	0.8	1.2	4.2	21.3	42.0	79.2	117.6
50	0.0	0.2	0.3	0.4	0.4	3.6	16.2	28.4	40.3	42.0
Mesothelioma In Male Nonsmokers										
0	0.1	0.6	1.0	1.6	2.2	12.5	57.0	2.3	164.5	220.1
10	0.1	0.4	0.6	1.0	1.2	7.8	35.3	2.6	97.3	122.6
20	0.0	0.2	0.4	0.5	0.6	4.5	20.4	5.1	52.4	61.7
30	0.0	0.1	0.2	0.2	0.3	2.4	10.5	7.5	24.6	26.9
50	0.0	0.0	0.0	0.0	0.0	0.4	1.5	2.2	2.7	2.7
Lung Cancer In Male Nonsmokers										
0	0.0	0.0	0.0	0.0	0.2	0.3	1.5	2.9	5.9	18.5
10	0.0	0.0	0.0	0.1	0.2	0.3	1.5	2.9	5.9	15.5
20	0.0	0.0	0.0	0.1	0.1	0.3	1.5	2.9	5.9	12.5
30	0.0	0.0	0.0	0.1	0.1	0.3	1.5	2.9	5.7	9.7
50	0.0	0.0	0.0	0.0	0.0	0.3	1.3	2.2	3.9	4.2

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[1] The 95 percent confidence limit on the risk values for lung cancer for an unstudied exposure circumstance is a factor of 10. The 95 percent confidence limit on the risk values for lung cancer on the average determined from 11 unit exposure risk studies is a factor of 2.5. The 95 percent confidence limit on the risk values for mesothelioma for an unstudied exposure circumstance is a factor of 20. The 95 percent confidence limit on the risk values for mesothelioma for a studied circumstance can be reasonably averaged as a factor of 5. The values for continuous exposure were derived by multiplying 40 hr/wk risks, obtained from occupational exposures, by 4.2 (the ratio of hours in a week to 40 hours).

Attachment 23-B

Table 23-B-1

Region I Population Data[1]

(1985 data, in thousands)

Region I State	Metropolitan	Nonmetropolitan	Total
Maine	420	744	1,164
New Hampshire	560	438	998
Vermont	122	413	535
Massachusetts	5,291	531	5,822
Rhode Island	896	72	968
Connecticut	2,940	233	3,173
Total	10,229	2,431	12,660

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[1] Source: U.S. Department of Commerce, Bureau of the Census, *Statistical Abstract of the United States*, 1987.

Table 23-B-2

Percentage of Males and Females in Each Region I State[1]

(1980 data)

Region I State	Percentage of Males	Percentage of Females
Maine	49%	51%
New Hampshire	49%	51%
Vermont	49%	51%
Massachusetts	48%	52%
Rhode Island	48%	52%
Connecticut	48%	52%

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[1] Source: 1987 *Statistical Abstract of the United States*. A breakdown of population by sex was available only for 1980; this breakdown was assumed to remain unchanged for 1985.

Table 23-B-3

Region I Population Data by Sex and Area[1]

(1985 data, in thousands)

Region I State	Metropolitan		Nonmetropolitan		
	Male	**Female**	**Male**	**Female**	**Total**
Maine	206	214	365	379	1,164
New Hampshire	274	286	215	223	998
Vermont	60	62	202	211	535
Massachusetts	2,540	2,751	255	276	5,822
Rhode Island	430	466	35	37	968
Connecticut	1,411	1,529	112	121	3,173
Total	4,921	5,308	1,184	1,247	12,660

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[1] Source: U.S. Department of Commerce, Bureau of the Census, *Statistical Abstract of the United States*, 1987.